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SOME POSSIBILITIES FOR MEASURING SELECTION INTENSITIES IN MAN[1]*

BY JAMES F. CROW
University of Wisconsin

IT is convenient to consider selection intensity at three levels—total, phenotypic, and genotypic.

There can be selection only if, through differential survival and fertility, individuals of one generation are differentially represented by progeny in succeeding generations. The extent to which this occurs is a measure of *total* selection intensity. It sets an upper limit on the amount of genetically effective selection.

Not all the differential can be associated with differences in phenotype, for there are large environmental and random elements in survival and reproduction. But, to the extent that differences can be associated with phenotype, *phenotypic* selection intensity can be measured.

Selection can be genetically effective only if it is *genotypic*, that is, if different genotypes make differential contributions to future genera-

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[1] Paper No. 660 from the Department of Genetics. I should like to acknowledge the help received in numerous discussions with Dr. Newton Morton and Dr. Ove Frydenberg.

* The first four articles in this issue were read before the Wenner-Gren Supper Conference on Natural Selection in Man, held at the University of Michigan on 12 April 1957. These papers are also being published as *Memoir No. 86* of the American Anthropological Association and also as a hard cover book, *Natural Selection in Man*, by the Wayne State University Press.

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tions. Thus natural selection is an inefficient process with only a fraction, perhaps a small fraction, of mortality and fertility differences being genetically effective. Furthermore, very little of the genotypic selection effects any permanent biological improvement. As many authors have pointed out (see especially Haldane, '54a), most of the selection is devoted to eliminating recurrent harmful mutants, maintaining systems of balanced polymorphism, and adjusting to momentary fluctuations in the environment—in other words, to maintaining the status quo. Selection is a necessary but not sufficient condition for the directional change of gene frequencies on which evolution depends.

The Total Selection Intensity [2]

The total selection that can occur in man is limited by his low reproductive rate, yet there is still room for fairly intense selection. The most fertile human populations produce 10 or more children per family. This means that if four-fifths of the children were to die prematurely the population number could still be maintained.

I suggest that the total amount of selection is best measured by the ratio of the variance in progeny number to the square of the mean number. (The parents and offspring are assumed to be counted at the same age, perhaps at birth.) This sets an upper limit on the amount of gene frequency change that selection can effect. The reason for the choice of this measure will now be developed.

I shall use *fitness* in a strictly Darwinian sense as the expected number of progeny, counted at the same age as the parents (Fisher, '30). In a precise theoretical analysis of populations, such as man, with overlapping generations it would be better to measure fitness as the natural logarithm of the progeny number, the Malthusian parameter of Fisher ('30: 25). But the cruder formulation is satisfactory for most purposes.

Suppose that the parental generation is counted at birth and consists of a fraction p_1 of individuals with fitness w_1, p_2 with fitness w_2, and so on $(p_1 + p_2 + p_3 + \cdots = 1)$. The average fitness of the population is given by

$$\bar{w} = \frac{\sum p_i w_i}{\sum p_i} = \sum p_i w_i.$$

Because of differential productivity, the expected frequency of type

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[2] This section has been greatly expanded since the oral presentation as a result of a suggestion of Dr. J. V. Neel, for which I am indeed grateful.

i next generation will be $p_i w_i$, assuming the complete heritability and constancy of fitness. The average fitness next generation will then be

$$\frac{\sum (p_i w_i)\, w_i}{\sum p_i w_i} = \frac{\sum p_i w_i^2}{\bar{w}}.$$

The relative increment in fitness between the two generations is

$$\frac{\Delta \bar{w}}{\bar{w}} = \frac{\sum p_i w_i^2 - \bar{w}^2}{\bar{w}^2} = \frac{V}{\bar{w}^2} = I \tag{1}$$

where V is the variance in fitness, and I will be called the *Index of Total Selection.*

This means that if fitness is completely heritable, that is, if each offspring has exactly the average of his parents' fitnesses, the fitness of the population will increase at rate I. A trait or a gene that is genetically correlated with fitness will increase in proportion to this correlation. The index therefore provides an upper limit to the rate of change by selection. The actual change in a character will depend also on its heritability and correlation with fitness.

It is of interest to separate I into two components, associated with mortality and differential fertility. This can be done as follows:

Assume p_d parents (counted at birth) die prematurely, and p_s survive until the child bearing period and have varying numbers of births $(p_s + p_d = 1)$.

			births (x)	
Premature deaths		p_d	0	$\sum_s p_i = p_s$
Survivals	p_s	p_0	0	$\bar{x}$ = overall mean number of births
		p_1	1	
		p_2	2	$\bar{x}_s$ = mean number of births per surviving parent
		p_n	n	$p_s = \bar{x}/\bar{x}_s$.

The index I, is defined as $V/\bar{x}^2$, where V is the variance in the number of progeny per parent (counting the non-survivors as leaving 0 descendants).

$$\begin{aligned} I &= \frac{V}{\bar{x}^2} = \frac{1}{\bar{x}^2}\left[p_d(0-\bar{x})^2 + \sum_s p_i(x_i-\bar{x})^2\right] \\ &= \frac{1}{\bar{x}^2}\left[p_d(0-\bar{x})^2 + p_s(\bar{x}_s-\bar{x})^2 + \sum_s p_i(x_i-\bar{x})^2 - p_s(\bar{x}_s-\bar{x})^2\right]. \end{aligned} \tag{2}$$

Now, let V_m be the variance due to differential mortality and V_f be that due to differential fertility.

$$V_m = p_d(0-\bar{x})^2 + p_s(\bar{x}_s-\bar{x})^2 = \bar{x}^2 p_d/p_s \tag{3}$$

$$V_f = \frac{1}{p_s}\sum_s p_i(x_i-\bar{x}_s)^2 = \frac{1}{p_s}[\sum_s p_i(x_i-\bar{x})^2 - p_s(\bar{x}_s-\bar{x})^2] \tag{4}$$

thus, from (2), (3) and (4)

$$I = \frac{V_m}{\bar{x}^2} + p_s\frac{V_f}{\bar{x}^2} = \frac{V_m}{\bar{x}^2} + \frac{1}{p_s}\frac{V_f}{\bar{x}_s^2} = I_m + \frac{1}{p_s}I_f \tag{5}$$

where

$$I_m (= V_m/\bar{x}^2 = p_d/p_s) \text{ and } I_f (= V_f/\bar{x}_s^2)$$

are the indices of total selection due respectively to mortality and fertility. The index due to mortality is especially simple, being the ratio of deaths to survivors.

Some numerical examples based on U. S. Census data are given in table 1. The 1950 and 1910 birth distributions are based on the total children ever born to all women who were of age 45-49 in the census year. The variance is somewhat uncertain because the Census reports lump several classes in the high birth numbers, but this is not likely to introduce a very large error. Another source of error is that no allowance was made for women who died during the childbearing period after having one or more children. It was thought better not to attempt such a correction, since there is almost certainly some lack of independence between fertility and mortality in these ages, and any correction based on an assumed independence might make matters worse. However, the error due to neglecting this correction is not likely to be large, for the death rates at these ages are very low, especially in recent years.

The three mortality values in table 1 are chosen to correspond roughly to the total mortality of women from birth to the end of the child bearing period for three time intervals: at present (10%), during the lifetime of women who were 45-49 in 1950 (30%), and during that of women who were 45-49 in 1910 (50%). Thus the most relevant comparison is between rows (2) and (6). During this 40 year period the index of total selection dropped from 2.6 to 2.1, or by about 20%.

It is interesting to note that the component of selection due to differential fertility actually increased during this period. Although the mean number of children per woman (including unmarried) dropped from 3.9 to 2.3, the index of selection due to fertility differences increased almost 60%. Despite a great lowering of fertility rates the pattern of marriages and births was such as to increase the effective fertility differential.

TABLE 1

The total intensity of selection, I, and its components for several hypothetical populations based on U. S. Census data

		I_m	I_f	I_f/p_s	I
(1)	1950 birth distribution, 10% mortality	.111	1.143	1.270	1.381
(2)	" " 30% "	.429	1.143	1.633	2.062
(3)	" " 50% "	1.000	1.143	2.286	3.286
(4)	1910 " " 10% "	.111	.784	.871	.982
(5)	" " 30% "	.429	.784	1.120	1.549
(6)	" " 50% "	1.000	.784	1.568	2.568

The first column, I_m, is the selection intensity due to mortality, the second column, I_f, is that due to differential fertility, and the last gives the total selection intensity from both causes. The third column gives the ratio of I_f to p_s the fraction of survivors.

The column I_m shows what the selective index would be if premature mortality were the only differential factor, *i. e.*, if all surviving women had the same number of children. This shows that with recent low death rates, differential post-natal mortality provides only a small part of the total opportunity for selection.

Measuring Phenotypic Selection Intensity

Haldane ('54b) has suggested a way of measuring phenotypic selection, and has used human birth weight as an example. He notes that infants of intermediate weight are more likely to survive the hazards of birth and early infancy than those at either extreme. Suppose that of those with the optimum weight a fraction s_o survive, whereas the overall survival is $\bar{s}$. Haldane defines the intensity of natural selection for birth weight as roughly $s_o - \bar{s}$, or more precisely, $\log_e(s_o/s)$. For example, where s_o is .983 and $\bar{s}$ is .959, the intensity of selection is $\log_e (.983/.959) = .024$. By Haldane's method, the intensity of selection for any quantitative phenotypic effect can in principle be measured. Since the subject is fully discussed in Haldane's paper, I shall say no more here.

Genotypic Selection Intensity: The Genetic Load of a Population

I should like to define as the *load* of a population the extent to which it is impaired by the fact that not all individuals in the population are of the optimum type. In the example above, the average survival is decreased by .024 from what it would be if all infants were of optimum weight.

What I wish to emphasize is the part of the population load that is due to genetic differences. The *genetic load* of a population is defined as the proportion by which the population fitness (or whatever other trait is being considered) is decreased in comparison with an optimum genotype.[3] Thus, if the character being measured is fitness, the genetic load is a measure of the total genotypic selection intensity.

The genetic load has many possible components of which I shall enumerate three that seem to me to be important and possibly amenable to study. For definiteness the discussion will be restricted to death rates, though other components of total selection intensity could in principle be studied this way.

1. *The mutation load.* Part, perhaps a large fraction, of the selection occurring in a population is directed toward the elimination of recurrent harmful mutants. The proportion by which the population fitness is thereby lowered is the mutational load.

2. *The segregation load.*[4] The optimum genotype might not be fixable. The simplest, and I suspect, most important example is a locus with a heterozygote favored over either homozygote with the population thus maintaining a balanced polymorphism. Inferior homozygotes are produced by segregation each generation, lowering the average population fitness and thereby creating a load.

The mutation and segregation loads, though difficult to separate in practice, are quite distinct in principle. Since mutation provides the raw material for evolution, the mutation load is, as Haldane ('37) notes, the price the species pays for the privilege of evolution. This load is independent of the system of heredity and would, for example, be the same in asexual as in sexual species. On the contrary, the segregation load could occur only in sexual species since it depends on Mendelian segregation.

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[3] It is quite likely that the optimum population is not made up of a single genotype, but of a variety. For example a society with division of labor would imply a heterogeneous optimum population. However, this possibility does not greatly change the essential problem, if one assumes that within each subset (*e. g.* males and females) there is an optimum genotype.

[4] Dobzhansky ('55, '57) has classified genetic loads into mutational and balanced. My segregation load is a part of his balanced load, since the latter also includes other possible types of polymorphism, *e. g.*, those due to factors that are favored in different niches, or are frequency-dependent. These latter types may not depend on segregation, and the method of analysis would be different.

3. *The incompatibility load.* This load comes not from the deficiency of any genotype itself, but from the fact that some genotypes have a reduced fitness with certain parents. For example, a zygote of blood group A seems to have a better chance of surviving if its mother is group A rather than group O. Such incompatibilities may possibly account for a substantial fraction of human embryonic deaths. Thus far, these are known only for serological characters, though later research may reveal other possibilities. I shall not discuss this topic further, since it is the subject of Levine's paper later in this symposium.

The Mutational Load

A quantitative assessment of the mutational load may be made by using a principle first discovered by Haldane ('37). Suppose the three genotypes AA, AA', and $A'A'$ survive and reproduce in the ratio $1:1-hs:1-s$.

Genotype	AA	AA'	$A'A'$
Frequency	p^2	$2pq$	q^2
Relative fitness	1	$1-hs$	$1-s$

Unless h is very small, the frequency of the gene A' in the population will be determined almost entirely by the fitness of the heterozygous class, for the homozygotes $A'A'$ will be so rare as to make only a tiny contribution to the selective pressure. Thus, we can ignore the $A'A'$ class and assume the population to be made up only of AA and AA' with negligible loss in accuracy. Every generation the AA' class will be reduced by a fraction hs because of selection, and at equilibrium this will be balanced by new mutations from AA to AA'. This leads to the equation:

$$(2pq)(hs) = m, \text{ or } 2pq = m/hs \tag{6}$$

where m is the mutation rate. This is the rate for both A genes or twice the rate per gene.

The mutational load, L_m, may be computed as follows. If there were no mutations the population would eventually consist only of AA genotypes with relative fitness 1. But the equilibrium population when mutation occurs consists of a fraction $2pq$ of AA' types, each of which lowers the fitness by hs. The total proportion by which the fitness is reduced, or the mutational load, then is

$$L_m = (2pq)(hs) = (m/hs)(hs) = m. \tag{7}$$

This illustrates the principle, which Haldane ('37) showed with less restricted assumptions, that the fitness of a population is lowered as a result of recurrent harmful mutations at a locus by a fraction equal to the mutation rate at that locus. The mutation rate, m, means the total rate for both members of a gene pair.

The significant point is that this is independent of the values of h and s, unless they are very small. If the mutations act independently, the total load becomes the total mutation rate for all loci. If h approaches 0, *i. e.* if the A' gene becomes completely recessive, the load becomes only half as large (since each death or failure to reproduce in a homozygote removes two deleterious genes). But there is reason to think from Drosophila data, and also from direct studies of a few genes in man (Muller, '50), that most "recessive" genes have enough of an effect in the heterozygous state that for the purposes of this analysis they may be treated as dominants.

The Segregation Load

I shall consider as the simplest example of a situation which leads to a segregation load a single locus where the heterozygote is favored over either homozygote.

Genotype	AA	AA'	$A'A'$
Frequency	p^2	$2pq$	q^2
Relative fitness	$1-s$	1	$1-t$

Such a population reaches a stable equilibrium determined by the relative values of s and t. In such a system, if s and t are large in comparison with the mutation rates, which is usually the case, mutation may safely be ignored. The equilibrium frequencies can be easily deduced by the following argument. For a more direct algebraic treatment see Wright ('31) or Fisher ('30 : 100).

Each generation a number of A genes proportional to sp^2 is eliminated because of the reduced fitness of the AA genotype. Likewise, a number of A' genes proportional to tq^2 is eliminated because of the inferior fitness of the $A'A'$ type. Whenever the eliminated proportion of both kinds of genes is the same, their ratio will not change, and the population will be in equilibrium. Thus, at equilibrium

$$\frac{p^2s}{p}=\frac{q^2t}{q}, \text{ or } \frac{p}{q}=\frac{t}{s}, \qquad p=\frac{t}{s+t}, \; q=\frac{s}{s+t}. \tag{8}$$

The proportion by which the average fitness of the population is reduced, in comparison with a hypothetical population composed of AA' heterozygotes, is given by sp^2+tq^2. If we substitute the equilibrium values of p and q into this, we get for the segregation load

$$L_s = sp^2 + ta^2 = st/(s+t). \tag{9}$$

Note that since s and t are ordinarily much larger than m—perhaps as much as two orders of magnitude in a typical case—the segregation load for a particular locus is much larger than the mutation load. But at the same time there must be many more loci of the type leading to a mutational load than those leading to a segregational, so that it is not possible from these considerations to say which has the greater total effect.

Separating the Mutation and Segregation Loads

In a human population these loads are usually indistinguishable. But there are two procedures which may enable some sort of separation of the two factors and make it possible to assess crudely their relative magnitude. The first depends on their differential response to inbreeding. The other is that the relative value of parent-offspring and sib correlations would be expected to be different for the two kinds of loads. These possibilities will now be briefly explored.

Consider a locus that is contributing to the mutational load, and assume that by some process the population is made completely homozygous but the gene frequencies stay the same as before. The population will then be composed of a fraction p of AA homozygotes and q of $A'A'$. Now, if p is nearly one, $2pq$ is almost twice the value of q. Therefore, by equation (6) q is approximately $m/2hs$. The load in such a hypothetical population is qs, or $m/2h$. Since the genetic load of such a locus in a randomly mating population is m, the ratio of the inbred load to the randomly mating load is $1/2h$. This principle has been used to provide an indirect measure of human mutation rates (Morton, Crow and Muller, '56).

Fortunately this ratio depends only on h, the amount of dominance, and not on s. There is no human information on h that is quantitatively reliable, but there is some information from Drosophila lethals and semilethals that h is about .02 (Morton, Crow and Muller, '56).[5] If this value holds for man, we should expect that the number of deaths in a completely inbred population would be about 25 times as great as for the corresponding category in a randomly mating population, or more if there is an appreciable fraction of complete recessives.

The situation is quite different for a locus where the polymorphism is maintained by the superior fitness of a heterozygote. In this case, if the population is made homozygous, the load becomes $sp + tq$. Substituting from (8) leads to a value of $2st/(s + t)$. Thus for a locus with a superior heterozygote, the segregational load is exactly twice as great in a homozygous population as in a randomly mating one.

If there are multiple alleles, the situation with the mutational load is unchanged; the value of m is simply the sum of the mutation rates to all mutant alleles. With the segregation load, multiple alleles complicate the relation.

Consider k alleles at a locus where all heterozygotes are equal in fitness and superior to any homozygote. If the homozygote A_iA_i is selectively inferior by a fraction s_i to the heterozygote, equilibrium is reached when $q_i = 1/s_i \sum(1/s_i)$ (Wright, '49: 372).

The load in a randomly mating population is $\sum s_iq_i^2 = 1/\sum(1/s_i)$. In a homozygous population the load is $\sum s_iq_i = k/\sum(1/s_i)$, or k times as great. When one heterozygote is more fit than the others, the ratio is less than k.

Therefore, unless the segregation load is based on loci with a large number of alleles, maintained in balanced polymorphism, it is not changed very much by inbreeding. In general, I would conclude that if the fitness is greatly decreased by inbreeding it is to that extent largely attributable to the mutation load rather than the segregational. This gives a possible basis for separation of the two.

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[5] The reader may be puzzled as to why this value of 2% is taken when most published data on dominance in Drosophila give the value as 4-5%. The reason is that for this purpose we want h for mutants in a natural population. Since those mutants with the largest h values are most rapidly eliminated, those remaining have a smaller average value of h. Recent unpublished results of Hiraizumi give a value of .024.

It is of course impossible to study completely homozygous humans. But it is possible by studying the children of consanguineous marriages of various degrees to infer what would happen with complete homozygosity. For example, one-sixteenth of the genes of the child of a cousin marriage are made homozygous as a consequence of the inbreeding, so the effect of complete homozygosity would be 16 times as great. Calculations (Morton, Crow and Muller, '56) based on the death rates of children from consanguineous marriages in rural France indicate that, whereas the average child from unrelated parents had a probability of about .12 of death between birth and sexual maturity, a complete homozygote would have the equivalent of about 2 lethal genes. Thus the inbreeding load is about 17 times as great as the random load. I conclude therefore that inbreeding effects are due largely to "ordinary" gene loci and not those maintained in polymorphic balance.

If we take the inbred load (assuming it to be entirely mutational in origin) and multiply by $2h$ we can estimate the mutational load in a randomly mating population. In this case the estimate is $2 \times .04$, or .08. This suggests that in this population .08 out of the total death rate of .12 is mutational in origin, which implies that at least $\frac{.08}{.12} = \frac{2}{3}$ of the deaths are genetically selective. However, the data are not very accurate and no one knows how well the average dominance agrees in Drosophila and man. But to the extent that the method and data can be trusted, it would appear that a substantial fraction of deaths between birth (including stillbirths) and sexual maturity owe their ultimate origin to mutation.

While on the subject of death rates of children of consanguineous marriages, I should like to call attention to the importance of measuring death rates in inbred individuals from primitive populations. The difference between consanguineous and non-consanguineous death and fertility rates in different populations would give a basis for assessing selection that is genetic in origin.

A second possibility for distinguishing between these two kinds of loads depends again on Haldane. Haldane ('49) noted that with a heterotic locus the parent offspring correlation in fitness is zero when the population is at equilibrium. On the other hand the sib correlation is high, approaching .5 for small values of s and t. The situation is quite different for a rare dominant factor. (For this purpose a "recessive" mutant having as much as 2% heterozygous effect would behave as a dominant in population dynamics.) In that case the parent-offspring and sib correlations would be about the same, approaching 0.5. Therefore, the extent to which the parent-offspring correlations in fitness agree with the sib correlations may be a measure of the extent of the mutational load in the populations.

This is much easier to discuss than it is to measure, largely for two reasons. First, these statements hold only for total fitness and not necessarily for its component parts. This would be especially troublesome, as Haldane points out, if there were a negative correlation between fitness components as, for example, if a low childhood survival rate were associated with high adult fertility. The other difficulty is separating genetic effects from environmental. A seemingly lower parent-offspring than sib correlation may mean only that the environments of the sibs are more similar. This might possibly be circumvented by using half sibs in place of parent-offspring correlations, if groups of full and half sibs in the same family could be found. Thus far the method does not appear too promising, but it offers some possibility.

Effect of a Changing Environment

Probably many genes are deleterious in any environment that is likely to be encountered, and others are uniformly beneficial. For such loci, environmental changes make relatively little difference. In contrast to these are genes or genotypes whose selective values are reversed with a change in the environment. It might at first appear that a change of environment that makes one genotype less favored while making another compensatingly more favored would not alter the average fitness of the population, but this is not so. The population is always worse off as a result of the change. The reason is that the newly advantageous genotype, not having had the benefit of increase by natural selection in the past, is rare, whereas the previously favored type is common.

Shifts of environment that cause such reversals of many loci place a load on the population that may be of considerable magnitude, and analogous to the mutational and segregational loads, though requiring different methods of approach.

SUMMARY

Selection intensity may be measured at three levels—total, phenotypic, and genotypic.

An *Index of Total Selection* is proposed, given by the ratio of the variance in number of progeny per parent to the square of the mean

number. The parents and offspring are assumed to be counted at the same age (say, at birth) and those that die before reproducing are regarded as having zero progeny. The index may be separated into components associated with mortality and differential fertility. Applied to U. S. Census data, the change from 1910 to 1950 was to a decreased opportunity for selection by mortality, but an increase in that due to differential birth rates.

Phenotypic selection is briefly discussed, using a procedure of Haldane's ('54b).

Genotypic selection is measured in terms of the *genetic load,* the extent to which the population is impaired by recurrent mutation (mutational load), by segregation from unfixable superior genotypes (segregation load), and by parental-child incompatibilities (incompatibility load) The mutation load is strictly proportional to the mutation rate, while the segregation load is almost independent of it. The mutation load is particularly sensitive to inbreeding, so the study of children of consanguineous marriages provides an opportunity for its measurement.

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THE INFLUENCE OF THE ABO SYSTEM ON Rh HEMOLYTIC DISEASE[1]

BY PHILIP LEVINE

Ortho Research Foundation, Raritan, New Jersey, and the
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IN the remarkable example of selection against the heterozygote Rh positive (*Dd*) infant suffering from hemolytic disease, one gene *D* and one gene *d* are eliminated in all fatal cases and in all surviving infants suffering the complications of kernicterus. With an incidence of genes *D* and *d* as 60 and 40% respectively in a Caucasoid population, there is a more rapid diminution of the less frequent gene *d*. It was quickly pointed out by Fisher *et al.* ('44) and Spencer ('47) that in families with loss through hemolytic disease there is a tendency to compensate by a larger number of pregnancies on the part of the Rh sensitized woman. This was shown by Glass ('50) to be true at least for his white population but not for Negroes, largely because of the larger number of pregnancies and larger number of surviving children per family in the latter group. In any event, compensation results in the survival of normal Rh negative (*dd*) and therefore unaffected infants if the husband is heterozygous. With each surviving child, two *d* genes are added to the population, and it is conceivable that for the white population the end result might actually be overcompensation. Even with homozygous husbands, an ever increasing number of severely affected infants are now salvaged.

This paper will deal with the various factors involved which play an important role in this example of selection against heterozygotes—those

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[1] The author wishes to acknowledge the help of Dr. Blandine Y. Gosselin in preparing the tables and the bibliography.

which may be classified as interfering with nature's experiment but particularly with the influence of ABO blood group systems.

A few remarks may be made regarding the incidence of hemolytic disease in general. In the first place, the incidence is directly dependent upon the number of Rh negative individuals in any given population (Levine, '42). Thus, Rh hemolytic disease is exceedingly rare in all populations that are almost exclusively Rh positive, such as Chinese, Japanese, and Amerindians. It is two to three times more frequent in white (15% Rh negative) than in Negroes (5 to 6% Rh negative). It should be more frequent in Basques who are characterized by 30% of Rh negatives, but a protective factor is the proportionately lower incidence of homozygote fathers. In a Caucasoid population the total incidence of Rh hemolytic disease is about one out of 150 full-term pregnancies. An important factor responsible for this comparatively high incidence of hemolytic disease was the random transfusions of the female population without taking into account the Rh differences (Levine and Waller, '46). With current and future transfusions of Rh negative girls and women exclusively with Rh negative blood, the incidence, particularly of severe and fatal cases, will be reduced to a considerable degree. Another factor interfering with nature's experiment of selection against the heterozygote is the successful therapy of severely affected infants and the almost complete reduction of kernicterus to its vanishing point (Hsia, Allen, Gellis and Diamond, '52).

Of all cases of hemolytic disease, about 12 to 15% end in stillbirths or hydrops with death shortly after birth (Walker and Murray '56). There is as yet no single therapeutic measure which will prevent stillbirths due to hemolytic disease. However, their incidence may be diminished somewhat if isoimmunization by random transfusions can be prevented so that the family is completed before stillbirths can manifest themselves.

The bulk of hemolytic disease is contributed by a comparatively small group of Rh negative women, but until recently there were no systematic studies to determine which of the Rh negative women would be capable of producing antibodies. In 1954, Owen, *et al.* invoked the phenomenon of acquired tolerance in order to explain the individual variations in the capacity of producing antibodies, advancing some evidence to indicate that Rh negative sensitized women were more likely to have Rh negative mothers than Rh positive mothers. The assumption was that in a pregnancy of an Rh positive mother with an Rh negative fetus, the latter is exposed to maternal Rh antigens and therefore will fail to produce antibodies as a result of acquired tolerance, just as in case of the multiple birth in cattle due to in-utero exchange of intact cells (Owen '45). This concept, however, failed to apply to a group of sensitized mothers who gave birth to infants with hemolytic disease, and similar negative results were previously reported by Booth, *et al.* ('53) in a series of 113 affected infants.

TABLE 1

The Possible Role of Acquired Tolerance

Rh STATUS OF THE Rh NEGATIVE MOTHER'S MOTHER

		Positive	Negative
No antibodies	(Owen, *et al.* 1954)	25	9
Antibodies	" " " "	3	14
Hemolytic Disease	" " " "	14	7
Hemolytic Disease	(Booth, *et al.*, 1953)	67	46

The concept of acquired tolerance in relationship to the facility of antibody production with subsequent hemolytic disease is indeed a fascinating one and requires a comprehensive systematic survey. But such studies must take into account the effect of ABO blood group compatibility or incompatibility as first pointed out by the writer in 1943 and amply confirmed by numerous investigators. A compatible mating may be defined as one in which the wife's serum if containing anti-A and/or anti-B will fail to react with her husband's red cells, while in an incompatible mating her serum will agglutinate her husband's red cells.

The original findings on the effect of ABO incompatibility in Rh sensitization and those in additional studies based on the largest number of matings are given in table 2.

In all series there is a lower incidence of ABO incompatible matings varying from 13 to 19.5% in the studies carried out after 1943 when more accurate criteria were available for selection of the cases. In the 3 series in which mothers previously transfused were excluded—those of Malone ('49), Donohue, *et al.* ('54), and Walsh and Kooptzoff ('54)—the values were 19, 18.4 and 14.8% respectively.

The author's series of 501 matings of husband and Rh negative sensitized wife are listed in table 3 as ABO compatible and ABO incompatible. In each case the blood group of the Rh positive husband is given first.

TABLE 2

MATINGS OF Rh POSITIVE HUSBANDS AND Rh NEGATIVE MOTHERS WITH AFFECTFD INFANTS

Author	Number of Matings	ABO Compatible	ABO Incompatible
Levine ('43)	random matings[a]	65	35
Donohue, *et al.* ('54)	random matings[b]	66.5	33.5
Levine ('43)	215	75.3	24.7
Van Loghem and Spaander ('48)	240	87	13
Wiener, Wexler, and Hurst ('49)	282	82.2	17.8
Malone[c] ('49)	373	81	19
Heisto[d] ('55)	507	81.5	18.5
Stratton ('53)	319	83	17
Donohue, *et al.*[c] ('54)	481	81.6	18.4
Race ('54)	247	84	16
Walsh and Kooptzoff[c] ('54)	216	85.2	14.8
Reepmaker ('55)	1742	81.5	18.5
Stern, *et al.* ('56)	238	87	13
Levine—present study	501	80.8	19.2

a. Based on an incidence of the following: group O 45%; group A 41%; group B 10%; group AB 4%.

b. Based on ABO grouping of 1,000 children over 6 months of age in the Hospital for Sick Children, Toronto.

c. Mothers giving histories of previous transfusions were excluded. In other studies no mention was made of transfused mothers. Such mothers were not excluded in the author's series of 1943 or 1954.

d. The incidence of group A in this series is 50.6 in contrast to 40% in other caucasoid populations (see table 6). The value of 18.5% incompatible matings in this population is to be compared with 40% incompatible random matings. This series includes most of the 185 matings previously reported by Brendomoen ('52), who reported 14% ABO incompatible combinations.

TABLE 3

THE 501 MATINGS WITH Rh NEGATIVE MOTHERS OF Rh AFFECTED INFANTS

ABO Compatible		ABO Incompatible	
♂ ♀		♂ ♀	
O × O	133	A × O	57
O × A	117	B × O	19
O × B	27	AB × O	0
O × AB	10		
A × A	100	AB × A	3
A × AB	6		
B × B	6	AB × B	1
B × AB	5		
AB × AB	1	A × B	5
		B × A	11
Total	405		96
Percent	80.8		19.2

TABLE 4

ABO RECIPROCAL MATINGS IN Rh HEMOLYTIC DISEASE

Selected From a Total of 3,123 Matings

	COMPATIBLE							INCOMPATIBLE					
♂ ♀	M *	R	H	L	Total Observed	Total Expected	♂ ♀	M	R	H	L	Total Observed	Total Expected
O × A	102	435	111	117	765	*764.0*	A × O	41	172	49	57	319	*320.0*
O × B	17	91	21	27	156	*167.0*	B × O	10	40	12	19	81	*70.0*
O × AB	10	29	10	10	59	*43.1*	AB × O	0	0	2 †	0	2	*18.0*
A × AB	4	19	19	6	48	*52.9*	AB × A	3	15	6	3	27	*22.1*
B × AB	0	5	0	5	10	*11.3*	AB × B	1	3	1	1	6	*4.7*
Total					1038	*1038.3*	Total					435	*434.8*

* M = Malone ('49)	373 matings
R = Reepmaker ('55)	1742 matings
H = Heisto ('55)	507 matings
L = Levine — present study	501 matings
Total	3,123 matings

† Dr. Otto Hartmann, Oslo, Norway, informed the author that further analysis of Heisto's series revealed that one mother was transfused prior to the delivery of the affected infant. In the second case the husband was in A_2B so that the fetal cells, if they were in A_2, would be poorly agglutinable and theoretically could survive in the maternal circulation.

The greatest discrepancy, however, involves the matings of O × AB and AB × O, and this becomes evident from the combined series of 4 studies listed in table 4 based on a study of a total of 3123 matings selected for Rh hemolytic diseases.

Aside from Heisto's series ('55), there are 49 O × AB matings but none of the incompatible AB × O.[2] It has been suggested by Reepmaker ('55) and Nevanlinna and Vainio ('56) that all group A and group B husbands in the incompatible matings are heterozygous and therefore of genotypes *AO* and *BO* respectively. Although genetically heterozygous, group AB husbands in the mating AB × O must be considered as homozygous in terms of ABO incompatibility because all infants must be in groups A and B and are necessarily incompatible with the group O mother.

Support for this view is found in the analysis of Nevanlinna and Vainio's data ('56) on 70 cases of hemolytic disease in ABO incompatible matings which included 15 group AB husbands. These 15 matings must be AB × A or AB × B, but AB × O should have been excluded because in each of his 4 classes to be discussed below, there were affected children compatible with their mother's blood group.

The higher incidence of compatible matings among Rh negative sensitized mothers of affected infants results in a distribution of their A-B-O blood groups which is different from that of their husbands or that of a random population. This is seen in the comparison of the blood groups in the author's more recent series of 1280 Rh negative mothers which include 501 husband-wife combinations.

The higher incidence of A among the Rh negative sensitized mothers, the lower group A and higher group O among their husbands was observed in all studies in which this analysis was made. In Heisto's Norwegian population ('55) characterized by an incidence of group A of 50.6% in a random population, the incidence of group A among the Rh negative sensitized women was 55.3. The differences of groups O and A among the Norwegian sensitized women and their husbands follow the same pattern as that seen in all other studies (table 6).

In both series shown in tables 5 and 6, the greatest discrepancy is seen in the incidence of group AB which is much higher in the mothers

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[2] In the authors series there were two AB × O matings but these were not included because in one the hemolytic disease preceded a remarriage with an AB husband and in the other the hemolytic disease resulted from a transfusion with a subsequently tested Rh positive donor.

TABLE 5

BLOOD GROUP DISTRIBUTION IN 1280 MOTHERS AND 501 MATINGS WITH HEMOLYTIC DISEASE

	1280 Mothers	501 Matings Mothers	501 Matings Fathers
	Percent		
O	41.4	41.7	57.3
A	47.0	46.1	33.5
B	7.5	7.8	8.2
AB	4.1	4.4	1.0

The 501 Rh negative mothers are included in the total of 1,280 mothers.

TABLE 6

INCIDENCE OF ABO IN NORWEGIAN POPULATION

(After Heisto, '55)

	11,644 random	745 Rh negative sensitized mothers	507 husbands of Rh sensitized women
O	37.9	30.9	46.5
A	50.6	55.3	46.2
B	7.8	8.5	5.5
AB	3.7	5.3	1.8

This series includes most of the 185 cases published by Brendemoen ('52).

than in their husbands. This is to be expected on the basis of the findings in the reciprocal matings involving an AB parent (table 4). A higher incidence of group AB mothers of Rh affected infants was previously observed by Chown ('48) and Davidsohn ('48).

As indicated above, the incidence of ABO incompatible matings in the three series of Rh hemolytic disease in which transfused mothers were excluded (Donohue *et al.*, '54; Malone, '49; Walsh and Kooptzoff, '54) did not differ much from those studies in which no mention is made of transfusion or in the author's series in which it was not possible to review the histories for this antigenic stimulus other than pregnancy. Possibly the role of previous transfusion in Rh hemolytic disease is now negligible because the current tendency is to transfuse Rh negative women or girls with Rh negative blood. However, Rh positive women or girls are as a rule transfused with Rh positive (or even Rh negative) blood irrespective of the difference in antigen c. During the period 1954 to

1956, 75 cases of hemolytic disease due to anti-c were studied in which blood groups of man and wife were available (Levine and Stroup). In 42 of these cases there was a history of previous transfusions, presumably with blood containing factor c. It is only after excluding these 42 cases that the protective effect of ABO incompatibility was noted in the 33 remaining cases and in these cases the incidence of ABO incompatible matings of 18.2 (table 7) did not differ much from the value of 19.5 in the author's series of Rh hemolytic disease shown in table 2.

TABLE 7

HEMOLYTIC DISEASE DUE TO ANTI-*c*

1954–1956

	ABO Incompatible Percent
Random matings	35
75 cases hemolytic disease	26.7
42 cases with transfusion history	33.3
33 cases no transfusion history	18.2

TABLE 8

MOTHER-CHILD COMBINATIONS

	Author	No. of Cases	ABO Incompatibility
Random, expected	Donohue, *et al.* (1954)		19.8
	Walsh and Kooptzoff (1954)		19.5
	Reepmaker (1955)		19.8
Random, observed	Wiener, *et al.* (1930)	1462	23.4
	Kirk, *et al.* (1955)	8055	18.1
	Lucia and Hunt (1950)	1167	19.8
Rh negative mothers of infants with hemolytic disease	Lucia and Hunt (1950)	120	4.4
	Donohue, *et al.* (1954)	451	7.8
	Walsh and Kooptzoff (1954)	222	11.7
	Reepmaker (1955)	1608	7.3
	Levine (this study)	293	12.6

[illegible] Research
[illegible]
Date ....

The effect of ABO incompatibility on Rh sensitization with resulting Rh hemolytic disease is seen also in mother-child combination. In contrast to an expected incidence of about 19% and values varying from 18 to 23% in observed mother-child combinations, the corresponding values in Rh hemolytic disease are from 4 to 12%. These relationships are tabulated in table 8.

In the series with hemolytic disease there is a distinct decrease of incompatible mother-child combinations, but there was a rather wide gap of 12.6 to 4.4 in the several studies. These variations could not be explained on the basis of immunization of the Rh negative mother by random transfusions, because Walsh and Kooptzoff who excluded these cases, report a value of 11.7 while the author, who excluded only those with transfusion histories, observed a value of 12.6.

TABLE 9

NORMAL AND Rh AFFECTED INFANTS IN 70 ABO INCOMPATIBLE MATINGS

(After Nevanlinna and Vainio, '56)

	NORMAL AND AFFECTED INFANTS	
	ABO compatible with mother	ABO incompatible with mother
Normal Infants		
Preceding last normal	17	12
Last healthy child	39	11
Affected Infants		
First	15	17
Others	15	16
Total	86	56

These discrepancies could probably be resolved in complete family studies which take into account the number of pregnancies, abortions, order of birth and the blood groups of all children, healthy and affected. This was in part undertaken by Nevanlinna (Nevanlinna and Vainio, '56), who limited his study to 70 ABO incompatible matings including 142 affected infants whose ABO blood groups were determined. (In 69 dead infants and 40 additional affected infants, blood tests were not available.) The ratio of the compatible and incompatible infants in the four classes are presented in table 9.

In this series the husband is necessarily heterozygous, as already suggested by Reepmaker ('55), so that the incidence of compatible and incompatible children to be expected is 50: 50. This value is disturbed only in the first two classes but mainly in group "last healthy child," in which the ratio of compatible to incompatible affected infants was 78: 22. These significant findings suggest that the essential antigenic stimulus is most frequently provided by an ABO group compatible Rh positive pregnancy resulting in a healthy infant or in an infant free from frank symptoms of hemolytic disease.

The question arises as to the source of the antigenic stimulus in the 11 ABO incompatible examples of "last healthy child" reported by Nevanlinna. Perhaps these mothers have received an intramuscular injection of blood not revealed in their histories, but it is more likely that the actual stimulus may have been provided by the "preceding" last healthy child who may have been ABO compatible with the mother. Other possible explanations will be referred to later.

TABLE 10

BLOOD GROUPS OF AFFECTED CHILDREN COMPARED WITH NORMAL

	Donohue, *et al.* ('54)		Reepmaker ('55)		Levine Affected Infants
	Affected Infants	Normal Infants	Affected Infants	Normal Population	
Group	451	1,000	1,608	68,000	293
O	51.2	46.9	50.4	45.3	49.1
A	39.2	39.9	40	42.9	39.6
B	6.7	9.7	7.5	8.6	8.2
AB	2.9	3.5	2.1	3.2	3.1

The distribution of the ABO blood groups of affected children shown in table 10 reveals a somewhat higher incidence of group O. This is to be expected if all husbands in the incompatible matings are heterozygous for A or B. The effect, however, is not striking because Nevanlinna's studies indicate that the greatest discrepancy in ABO blood group distribution is to be expected in the class "last healthy child" rather than in the affected infants. The main source of the excess of group O affected infants is derived from the high incidence of compatible O × O and O × AO matings.

The most plausible explanation for the failure of Rh positive, ABO incompatible pregnancies to provide a suitable antigenic stimulus is the intravascular destruction of the group incompatible fetal Rh positive

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red cells by maternal antibodies so that they do not reach the sites of antibody production in the mother. Some support for this view is provided by the results of experiments by Stern, *et al.* ('56), who injected group compatible and group incompatible Rh positive blood into volunteer Rh negative recipients. The striking difference in the antigenic response in these two series is seen from the summary of the results in table 11.

TABLE 11

39 Rh NEGATIVE VOLUNTEERS INJECTED WITH ABO Rh POSITIVE BLOOD

(After Stern, *et al.* '56)

		Range Titer 1:	Median Titer 1:
Compatible	10/17	16-512	128-256
Incompatible	2/22	2-8	2-8

After 4 intravenous injections, 10 of the 17 compatible group produced antibodies, while only one among the incompatible group produced antibodies. The second of the latter group developed antibodies after a fifth injection.

These significant findings indicate that Rh antibodies are produced far more readily and to a higher titer following the antigenic stimulus of group compatible blood as compared with injection of group incompatible blood. Apparently, survival of the injected Rh positive red cells in the Rh negative recipient is essential for isoimmunization. As to the two of the 22 individuals who produced low-titered Rh antibodies following injection of ABO incompatible blood (table 11), one may assume that some Rh antigenicity may have resulted from slow destruction perhaps due to the combination of poor agglutinability of the red cells and low titered isoantibodies in the recipient's serum. Survival of red cells, however, is not the only explanation because in the compatible group 7 failed to produce antibodies, and among the 10 who did produce antibodies, some responded more readily than others as indicated by the first appearance of antibodies after a variable number of injections and by varying range of titers. As pointed out by Owen, *et al.* ('54) it would be interesting to determine whether or not there is a higher incidence of Rh positives among the mothers of the 7 resistant or tolerant recipients.

By analogy, one may state that in contrast to an ABO incompatible pregnancy, a compatible pregnancy provides favorable conditions for survival of the Rh positive fetal blood in the Rh negative mother. Among the ABO compatible pregnancies, however, there are two strikingly different sorts of obstetrical histories. In one—by far the smaller group—the first Rh positive infant is normal or even mildly affected and the second and all other Rh positive infants are severely affected. In the contrasting and by far the larger (control) group, the Rh negative mother delivers two, three or more ABO group compatible Rh positive infants free from hemolytic disease. (In both classes, those giving clear-cut histories of transfusions of Rh positive blood or abortions should be excluded.) In this comparative study the mother's parity, order of birth, and birth rank of the first affected child should be recorded and compared with the control group of Rh negative women of equal parity who fail to produce antibodies. With this selected family material—limited to ABO compatible pregnancies—it should be possible to determine the role of acquired tolerance, if any, or lack of genetic capacity to produce antibodies in order to explain the absence of hemolytic disease in ABO compatible pregnancies. A high incidence of Rh positive mothers of Rh negative daughters should be associated with tolerance or little if any hemolytic disease, while the combination of Rh negative mother and daughter should yield many more examples of hemolytic disease. If, under the latter conditions, hemolytic disease does not appear, one may assume a lack of the genetic capacity to produce antibodies.

The production of Rh antibodies in the face of a group incompatible pregnancy is an unlikely event unless the infant's Rh positive red cells manage for one or another reason to survive long enough to provide an antigenic stimulus, *e. g.* a very low titer of anti-A or anti-B in the maternal serum and poor agglutinability of the fetal red cells. At this point one may also mention another set of circumstances which would permit survival of group incompatible fetal red cells of normal sensitivity. This deals with another example of possible acquired tolerance if the Rh negative women, *e. g.* those of group O with group A husbands, were daughters of group A mothers and thus acquired tolerance manifested by failure to produce anti-A when stimulated either by group A offspring or injected with group A red blood cells or A soluble substance.

The findings presented may have a bearing on the mechanism of transplacental isoimmunization. If survival of fetal red cells in the maternal circulation is a condition for isoimmunization, this would suggest that it is the intact fetal red cells which find their way into the maternal circulation either during the course of the pregnancy (Levine, '43, '44) or at the delivery (Davidsohn, '45) of the "last healthy"

ABO group compatible child. It is assumed that this passage of fetal blood into the maternal circulation occurs in all pregnancies and that the failure on the part of many Rh negative women to produce antibodies as a result of group compatible pregnancies in the absence of acquired tolerance is determined genetically.

It has been calculated by Reepmaker ('55) that in the absence of protective effects of ABO group incompatibility, there would be an increase of 23 % in the incidence of Rh hemolytic disease. Incidentally, these findings offer a basis for better prognosis in future pregnancies on the part of Rh negative nonimmunized mothers of ABO group incompatible matings. The prognosis is still better in the specific mating AB Rh positive husband × O Rh negative mother. This aspect will be discussed in future publications.

SUMMARY AND CONCLUSION

In any discussion as to the fate of gene *d* in selection and the effect of compensation, or the possible role of acquired tolerance, it is now evident that one must take into account the protective role of ABO incompatibility in reducing the incidence of Rh hemolytic disease.

ADDENDUM

In the series of Owen, *et al.* ('54) there is an indication that acquired tolerance may possibly play a role in the class listed in table 1 as "no antibodies" after three Rh positive pregnancies since the disproportion of positive and negative maternal grandmothers is considerably greater than in the series of hemolytic disease. In the 17 cases of antibodies but no hemolytic disease, however, the next pregnancy with an Rh positive fetus would certainly result in an affected infant. The findings in these two groups could be explained also if the "no antibodies" class had a high incidence of ABO incompatible matings while the "antibodies" group had a high incidence of ABO compatible matings.

The findings presented provide substantial evidence that the intact group compatible fetal red cells find their way into the maternal circulation. More direct evidence was supplied by Dr. A. Richardson-Jones (oral communication) who with the aid of a sensitive technic for the detection of a very small number of Rh positive red cells in a mass of Rh negative red cells demonstrated fetal Rh positive cells in the circulation of the Rh negative mother. This important observation was made in Rh negative mothers tested during the last trimester of their pregnancies. Dr. Richardson-Jones estimates a ratio of 1:2000 — 1:5000 fetal red cells to maternal red cells.

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AN ANTHROPOGEOGRAPHIC EXCURSION AROUND THE WORLD[1]

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UNDER the aegides of the University Museum of the University of Pennsylvania, the Wenner-Gren Foundation for Anthropological Research, Incorporated, the Research Institute of the United States Air Force, and assisted also by *LIFE* magazine, my wife and I made a trip around the world, westward, taking 166 days. We left home on October 15, 1956, and returned on March 29, 1957, having visited en route Japan, Korea, Formosa, the Philippines, India, Nepal, Ceylon, Pakistan, Saudi Arabia, Italy, and France. In each country mentioned an effort was made to call on local anthropologists or cognate scholars, to see as much country as possible, to study skeletal and archaeological collections, and to look at people. Over 4,000 photographs were taken, about equally divided between black-and-white and color.

Moving about fairly rapidly, although comfortably, in heat and cold, and at various altitudes, and being plied with hospitality in all quarters, we saw and heard more than could be immediately digested, and it has not all been assimilated yet. This account is not a scientific document, nor even a contemporary one in its feeling and spirit; it is meant to be more like the travel notebooks produced by naturalists in the more leisurely and less sophisticated days of a century ago, when the world was still full of wonder, the *National Geographic* had not yet been invented, and a man could still interest himself in every living phenomenon without appearing naive or stepping on someone else's tender toes.

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[1] On April 12, 1957, as a participant in the Wenner-Gren conference on Natural Selection in Man, held at Ann Arbor under the leadership of Professor James N. Spuhler of the University of Michigan, I gave an impromptu talk on my recent trip around the world, using a set of color slides as framework. This was not a "paper" in any sense of the word, as no paper was involved in it outside the slide mounts. This written effort is a five-months late post-mortem attempt at recapitulation.

After landing at the Tokyo airport, our first stop was the International House of Japan, where we became the guests of Gordon and Jane Bowles. For over three weeks Gordon and I were able to renew our old association, and discuss particularly the many problems of human biology in Japan, where he was born and where he has studied and taught at various intervals over his entire life as a scholar. From him and from his Japanese associates [2] I learned as much as I could absorb in the time available. Together we went Ainu-watching on Hokkaido, and also visited the Kyoto-Nara-Tenri area; we also examined the famous Ubayama shellheaps and visited Father Marringer's mission museum. Under other guidance we visited the mountain country back of Kyoto and the fishing villages outside of Osaka.

Japan is often divided into three main geographical provinces with cultural overtones; Old Japan is the Inland Sea-Nara-Kyoto-Osaka region, comprising southern Honshu west of Lake Biwa and the two islands of Kyushu and Shikoku; New Japan is the rest of Honshu including the Tokyo-Yokohama area; the third province is Hokkaido. The "New Japan" part of Honshu can be further divided into the warm eastern coastal plain, the mountain core, and the cold, snowy west coast. Each of the three major areas, and three minor ones, has its distinct anthropogeographical problems.

During at least one of the glacial advances of the Pleistocene, the three southern islands were without reasonable doubt accessible from the mainland, to man as to other fauna. There is no reason therefore why archaeological remains comparable to those from the Choukoutien and other mainland Chinese sites should not be found, and fossil man as well. That they have not is probably due mostly to a paucity of interest. So firmly has the Japanese origin myth been imbedded in the minds of the people that to look for something older than the first man would, until the Emperor's recent voluntary demotion from godhead, have been, if not sacrilegious, at least in poor taste. In gravels in the Tokyo region, Father Marringer and the Meiji University staff have actually found flake implements typologically comparable to industries on the mainland and in southeast Asia, but their exact age is not yet known. Father Marringer's own feeling ('56 : 175-193) is that they are immediately post-glacial and represent a survival in vacuo of a much earlier industry. With Gordon Bowles we visited a Mesolithic site at Asamushi, near Sendai in Northern Honshu, where we collected a few pieces of a locally well known pre-pottery industry known as Iwa Juku, after its type site near Tokyo. After Iwa Juku comes the famous ceramic Jomon culture, the first industry to yield human remains. These have now been found from all periods. I had an opportunity to examine many of them at Kyoto. Although variable, they are definitely not Ainu, in the sense of the present Ainu of Hokkaido, nor are they very Mongoloid. The Jomon people, concentrated along the coasts where they ate quantities of shellfish, resembled cranially the predominant population element of the coastal fisherman today; they were brachycephalic and more Alpine than Mongoloid-looking. The heavily Mongoloid element apparently first arrived with the invasion, from Korea, of the Iron Age rice-cultivating Yayoi people, some of whom were also long faced and convex-nosed. From time to time other Koreans have entered Japan as craftsmen and been absorbed.

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[2] Thes included Professor Y. Iwamura, J. Kawakita, S, Kodama, T. Mabuchi, M. Oka, K. Segawa, A. Suda, S. Sukihara and H. Suzuki.

If one looks at Japan in terms of the mountains-coast dichotomy, it is clear that the most Mongoloid people live in the mountains, that the fishermen of the coast are predominantly Alpine, and that the city people have developed a type of their own which is brachycephalic and long faced, with relatively little Mongoloid appearance in the anatomical structure of the malars and nose. Various explanations were given me by my learned amanuenses; that the isolation of upland villages and villages on the west coast have so fostered the growth of local populations that many new types have arisen; that there has also been a continuous selection of the sedentes-migrants character by which the narrow faced people have been drawn to the cities (Shapiro and Hulse, '39; for the I. Q. aspect of a similar situation, see Scott, '57 : 311-329), and that upper class Japanese have deliberately bred a Samurai-looking type from second marriages with geishas.

TABLE 1

	SEX	AGE	STATUS
NARROW FACE	Male	Old	Higher
BROAD FACE	Female	Young	Lower

The narrow faced condition, along with light skin color, is definitely the more desired and sought after. By "seeking after" we include the use of artifice; cosmetic surgery to build up the nose and remove epicanthic folds is a thriving business in Japan; so is the manufacture of skin-bleaching substances, and of false breasts and buttocks. While at present there may be an element of imitation of the Caucasoid ideal in the motivation of these steps, the great motivation antedates our own (American) brief period of being chic, and goes back to the ancient Samurai ideal, in fact to the inspiration of the horse-faced and horse-riding invaders from whom the nobler Japanese are believed to be more largely descended than their commoner fellow-citizens. It is my own feeling that, all else equal, a gerontomorphic Japanese, to use the younger Marett's word ('36), is somehow better suited by temperament to endure the crowded life of a Japanese city than his moon-faced cousin, and that we have a principle here which can stand exploration in other countries. If I am right, then Professor Suzuki's dilemma, of how the embodiments of his mesocephalic and platyrrhine Mediaeval Japanese skulls sired the modern brachycephalic and leptorrhine urban Japanese, may be on its way to a solution.

Hokkaido is believed to be a separate anthropological province because the straits which separate it from Honshu are too deep to have been dry in glacial times, and because the Japanese still feel an even greater aversion to the idea of being descended from the hairy and dirty Ainus than they (until recently) did to finding a man older than the sun goddess's offspring. Despite a body of linguistic evidence which carries Ainu place names as far south as Lake Biwa, and the historical account of how the Japanese drove the Ainu north out of Honshu, and despite the patent antiquity of the Ainu in Hokkaido, most Japanese scholars now claim that they were recent arrivals from the north, in fact not much earlier on the spot than the Japanese themselves. One reason is the lack of Ainu skeletal material more than 300 years old. However, skeletal material is scarce in all countries where the ground is frozen many months of the year. Ainu burial customs do not promote preservation.

During the last three years archaeologists from first Meiji and then Hokkaido University have been digging sites along the northern shore of the island, and turning up amazing materials. In a number of separate sites, distinguishable in terms of soil type and depth but not by mutual stratigraphy, they have found (1) an assemblage of large obsidian blades with carinated steep scrapers, similar to my Kara Kamar III material from northern Afghanistan, there dated at more than 34,000 years old (Coon and Ralph '55: 921-922); (2) a similar assemblage but with finer blades, and including burins; (3) an even finer assemblage of blades, and with burins and projectile-heads, the latter retouched part way across the back with what seems to have been pressure-flaking; (4) microliths; (5) a kind of arrowhead resembling the North American "woodlands," with pottery.[3]

Not only is the sequence still tentative, but the C-14 dates are not yet in. In any case, these interesting discoveries show that Hokkaido was at one time in the same general archaeological world as north central Asia, eastern Siberia, and early North America. Whether or not its evolved Stone Age cultures extended across the stormy but narrow waters of the Tsugaru Channel remains to be discovered. As not a single pre-pottery cave site has yet been dug in Japan, almost anything can turn up.

Returning to Hokkaido, we find that about 1,000 years ago a people called Moyoro invaded Hokkaido from the north, bearing ivory and bone implements with flint points set into the bone; their culture was more or less of the Bering Sea type. Being entirely coastal, they settled at Abashiri, where some of their skulls have been dug up. These are big, heavy, and Eskimoid. The Jomon people also got to Hokkaido, but in quite late Iron Age times, and some of their skulls are available for inspection in Professor Kodama's museum. These are very variable, not simply, or even predominantly, Alpine as in the earlier southern collections, but more Mongoloid and in some cases beaky-nosed.

With all this going on in Hokkaido it is not to be wondered at if the local Ainu are not all of one piece, nor should we be surprised, if we ever find Ainu remains from Honshu, if they should turn out to be different from the living and recently dead Ainu of Hokkaido. However, when one surveys Professor Kodama's vast array of a thousand Ainu skulls, several things meet the eye at once. Although big skulls, of majestic architecture, they look less like modern European skulls than the living races resemble each other. This struck me all the more forcefully because the day before seeing them I had stood next to an Ainu of my own age and except for his greater interorbital distance and lower nasal bridge, we might have been brothers.

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[3] In his 1956 excavations Professor Kodama was accompanied by an American Fulbright botanist, Mr. Theodore Bank.

They are more massive than most European skulls; the canine fossa is lacking, and the teeth are larger. Despite the wholly Caucasoid orbit, there is a faint Mongoloid look that defies pinpointing, except in the fact that many of the Ainu teeth are shovelled, without, however, being procumbent as in many Japanese skulls. Furthermore, according to Professor Kodama, the Ainu teeth have an incidence of 30% of enamel pearls, a condition rare in other populations, except for the Eskimos, and one indicating a considerable genetic isolation.

It is Professor Kodama's stated opinion that the Ainu skull falls closest to the pre-Neanderthal specimens from Europe, such as Krapina, rather than resembling the Upper Paleolithic Cro-Magnons, and that the Ainu are thus an exceedingly ancient and primitive Caucasoid race; this is borne out, he says, by the neuroanatomy of the Ainu motor area, which is comparatively simple. While Ainu brains are larger than those of the Japanese, he said, they are not as complexly organized. Professor Kodama made his vast reputation not by collecting skulls or digging up flints, but in neuroanatomy. I have a lively respect for both his erudition and his wisdom. The problem of Ainu origins has not yet been solved, but the solution is on its way.

From Japan we went to Korea as guests of the United States Air Force and of the McGraw Hydrocarbon Company at Chungjo. With a recently returned Harvard graduate student, Mr. Kim Jong Hak, we explored a cave at Puk-Ha-Ri, just north of Tanyung. As no prehistoric archaeology had been done at all in Korea, except for some shell-mound work on offshore islands, it seemed a good idea. Caves are rare in Korea; the limestone is extremely dense and hard, and has few solution cavities. This cave is ideally situated above a river, and indeed, has a spring inside on one side; the walls slope outward and downward, and the fill is soft yellow earth. With local help we went down 80 cm through sterile earth to what seemed to be Neolithic material, including one typical celt-sharpening stone. I was and still am haunted by the idea that Sinanthropous remains must be just as abundant in Korea, where we can go, as in China, where we cannot. Future work will, I hope, tell whether Puk-Ha-Ri is the right site or not.

Compared to Japan, the racial situation in Korea seems fairly simple. A stature-weight cline undoubtedly exists from north to south, following Bergmann's rule. Head and face form are relatively uniform, with extreme brachycephaly combined with long faces and narrow noses; at least part of the Japanese ideal seems to have its prototype here. My feeling was that the Koreans are a kind of Turk; I was reminded of the Turkomans of the Gurgan Plain in Iran not only by their faces but also by the common occurrence of lean body build, and the occasional green eye and blond mustache hair.

In Seoul we were most graciously received by President Yun Il Sun of Seoul National University; he introduced us to Dr. Rha Sea-in, head of the Anatomy Department, and Dr. Han Pumsuck, the director of the Public Health School. With these two medical men I went into the question of the anatomy of the Mongoloid face; it is very simple, they said, and showed me that the key to the whole complex is the position of the temporal muscles, which are attached farther forward on the frontal bones than they are in Caucasoid skulls. In the area behind the orbit they press farther forward than they do in the Caucasoid skull, and reduce the depth of the orbit, pushing the eyeball forward in its socket. The Mongoloid masseters are also attached farther forward than in the Caucasoid skull. The whole Mongoloid mandibular musculature is hung relatively far to the front. The nasal skeleton also differs in several ways, being more built-in than the Caucasoid apparatus and forcing the eyeballs farther apart. Later on I learned from a noted

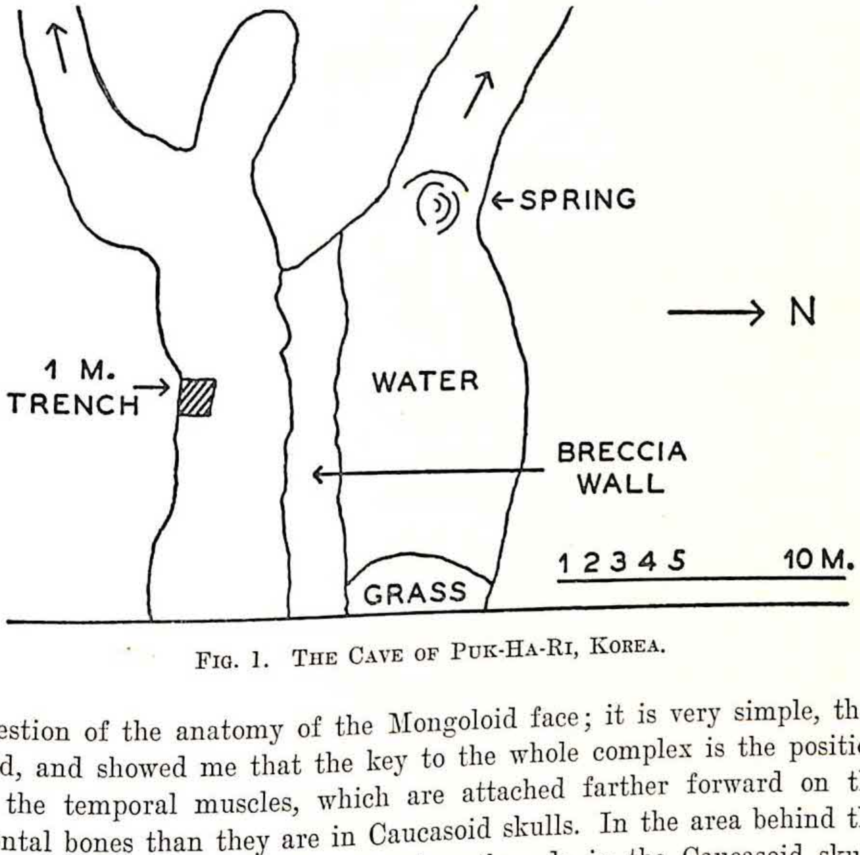

Fig. 1. The Cave of Puk-Ha-Ri, Korea.

eye-surgeon in Shillong, India, Dr. B. G. Guha (the anthropologist's brother), who operated daily on both Caucasoid and Mongoloid eyes (Khasis, Bengalis, and Europeans), that of the two the Mongoloid eyeball is the smaller. After seeing a number of Korean-American hybrids, who could pass for Americans, I came to suspect that the genetic basis for North European-Mongoloid differentiation, at least as far as the facial structure is concerned, is small and simply organized.

After returning to Japan we flew to Formosa, where we were warmly greeted by Professor Li Chi, director of the Academia Sinica and head of the Anthropology Department at the University of Taipei. He showed us his material from the Bronze Age site of An Yang, which, owing to the vicissitudes of war, he has had to move 14 times. He also let me see the famous An Yang skulls, and measure 8 of them. These had once been measured by Professor T. L. Woo, whose present whereabouts, if he is still alive, are unknown. These skulls, which came from sacrificial burial pits, seemed to be mostly or entirely female. Three distinct types were present; a dolichocephalic, modern North Chinese type; a heavy, brachycephalic, very Mongoloid form, and two skulls that could have been North Europeans, except that they had shovel incisors. The principal features of these two that distinguished them from the others were deep orbits and large prominent, nasal bones. However, that does not mean that their soft parts were Caucasoid, or that similar skulls could not be found among the living North Chinese population. This extremely significant series may well show, when finally studied and published, that at the very beginning of Chinese history the population of the North China plain was variable, and probably of multiple origin.

Going south to Taichung with our Assistant Air Attaché and host, Capt. Arthur Prager, we stayed with old State Department friends, Robert and Carol Lindquist, then attending the State Department Foreign language school. With them and others we made daily excursions into the mountains to see and photograph the aborigines, concentrating on the Atayals, a nation of 38,000 people who occupy a fifth of the island, less than half of which is inhabited by ten million Chinese. Head hunting helped keep the invaders at bay for centuries; now they are kept out by government decree and the fact that the aboriginal territory is too steep for rice terracing except at enormous effort. As in Japan and elsewhere, rice, both master and slave of man, feeds tens of millions while crowding them into limited areas.

The Atayals are physically variable. Some of the older women could be taken easily for Ainus; the tattooed whisker, reaching from lip to ear, heightened the natural resemblance. Two women whom we saw could easily have been Italian; a third Maya Indian. Heavy browridges and deep-set eyes characterize many of the men. Traces of blondism of hair and eye, with fair skins, are common. In contrast to the Atayals, the other two tribes whose members we saw, the Thaos and Bununs, are brown-skinned Alpo-Mongoloid people closely resembling the pagan tribes of Luzon to which they are culturally related. The racial history of Formosa promises to be no simple web to unravel.

Another hop took us to the Philippines, for a mere 4 days; one was spent visiting the Negritos at Clark Field with Dr. William T. Ward of the Air Force research staff, and the Crow Valley tribe with Captain J. A. Simon, who took us there in a Cub airplane. Although uniform in stature, the Negritos vary enormously in other respects; one old lady could have been a Bushman, and one man looked exactly like a miniature replica of a huge Australian aborigine named Tuki whom I measured in Melville Island in 1954. Hybrids were all above the standard 150 cm level.

Next we visited Manila, where the dean of Philippine anthropology, Professor Otley Beyer, very graciously gave us two full days of lecturing, demonstration, and wisdom. In his mature opinion, no cultural change took place in early post-Pleistocene times to warrant the idea that Negritos invaded the island with a Mesolithic culture; as other pygmy fauna have been found there, he suggests that the Negritos are the descendants of earlier, full-sized inhabitants, reduced by some selectively favorable genetic agency. Quite independently he worked out the same hypothesis arrived at by Hiernaux, Adé, Gusinde, and others for the African pygmy, and by Tobias for the Bushman. It is by far the simplest and most plausible hypothesis available. He also told us and showed us many other things, which greatly enhanced my education on the subject of these islands and their inhabitants.

A son, daughter-in-law, and 4 grandchildren awaited us in New Delhi. Eighty-five of our 166 days were spent in India, 8 in Nepal, and 6 in Ceylon. These 99 days were among the most profitable of my life. We first went to Jaipur and out onto the Thar Desert, to see how deserts in India compare to others elsewhere; then to Ranchi, where we stayed with my old friend and former teacher, Professor B. S. Guha, once head of the Department of Anthropology of the Government of India, and now director of the Bihar State Tribal Institute at Ranchi.

He took us about among the Chota Nagpur Hills, introducing us to many kinds of aboriginal people, the Oraons, Ho, Santal, Munda, Asuras, and Birhors. With us he went to Gauhati in Assam, and up into the Khasi Hills, to Shillong and Cherrapunji. Although unable to accompany us further he directed us from his driver's seat at Ranchi. In and about Cochin we saw the dwarfed Kadars of the forests, White Jews, and Malayalams; also Lake Periyar and the game reserve, Tamil country. We went with an Indian Buddhist mountaineer, R. N. Rahul, to Darjeeling and Kalimpong, and called on the Tibetan scholar, Georges de Roerich; then with Prince Peter of Greece to the Bhutanese lamasery of Pedong, and the Pedong fair; then to Kathmandu, where with John Pelzel of Harvard we joined Colin Rosser of the London School of Economics in a visit to his study-spot, the small town of Sankhu, an unspoiled gem. We also visited the great linguist, Colonel T. W. Clark, at Kathmandu. Later we went to Ceylon where we were shown a collection of new skulls and archaeological materials by the naturalist Paul Deraniyagala, director of the National Museum, and were given an entree to visit the Veddas by their special caretaker, Dr. Richard Spittle. Only three days were left for parts of Pakistan, which we had seen before.

India—here I use the word geographically only, for it cannot be divided by anyone but politicians—is a world in miniature. It has every known climate except tundra, and every known major race of mankind except the Bushman. Its people live in every known level of social complexity, from that of the simplest bands of root-diggers to that of the world's greatest statistician—Mahalanobis,—and of philosophers of the stature of Rabindranath Tagore. In one place or another and in one way or another, India has nearly everything. After 52 years of hearing and reading about India I should have known this, but it took a personal visit to have the impact hit me between the eyes. Either India is the home of all peoples, or the place where they all overlap, or, more likely, a combination of both.

Of the many tribes of simple food gatherers, I saw but three; the Birhors, Kadars, and Veddas. The Birhors, who live by catching monkeys in nets, digging roots, and trading rope for manufactured products, are small but not dwarfed people of an essentially Caucasoid body habitus and facial configuration. They must be among the oldest inhabitants of India, pre-Munda and pre-Dravidian, although they speak a form of Munda. The Kadars, who are root-diggers and honey-collectors, live the simplest possible kind of life. Before obtaining iron tools in trade they used only a digging stick, tipped with a roughly flaked stone. Some of the Kadars whom we saw in a single community were essentially Negritos; others looked Veddoid, or Australoid, or in between. The Veddas, who hunt with the bow (the Birhors and Kadars do not usually use it) are like the Birhors, miniature Caucasoids of a very basic and variable kind.

After I had seen these three tribes it came to me why the Dravidians and Aryans had been able to adapt themselves to the miserable heat of the Indian plains; they absorbed, seriatim, remnants of these earlier peoples of the same general strain as themselves, peoples whose adaptation to the terrain went back to the Pleistocene. Thus they survived, as the Anglo-Indians have in turn, while the full-blooded British have departed. The capacity for survival is not only a matter of climatic protection, but of neurological adaptation as well, which is a broad term embracing cultural phenomena.

Then it occurred to me why the Mongoloids had been able to live in the heat of Indo-China, Burma, and Siam—because they had been preceded in the steaming forests by earlier Mongoloids, the People of the Yellow Leaves, who had been able to transfer their genetic basis for survival without a loss of essential Mongoloidism. Many Mongoloids have come into India at one time or another, with different results.

In Calcutta I was privileged to examine certain still unpublished skulls from the Indus Valley civilization and this was as massive a milestone as the examination of the An Yang skulls in Taipei. These were equally variable, including some oversized Caucasoids that might have come from Sumeria or Denmark, some gracile Mediterranean crania, one patent Australoid, and one full Mongoloid, as out of place in the Indus valley in the third millenium B. C. as he would be today. Another principle was indicated, that multiplicity of genetic origin is an ancient thing in civilized places, while the mowing hand of natural selection cuts the originally variable grain to standard sizes. There were no autochthonous Mongoloids in India to do the job of the ancestors of the Birhors, Kadars, and Veddas.

When we moved into the Chota Nagpur hills, three kinds of people were in evidence: (1) The Caucasoid Birhors, who spoke a language probably not their own; (2) the Australoid Oraons, speaking Dravidian; (3) the phenotypically heterogenous Munda-speaking peoples, including the Munda proper, Ho, Santal, and Asuras. Their languages are related to Mon, Khmer, and other forms of speech native to southeast Asia, whence their early food plants and shifting agriculture seem to have come. In a nutshell, the men are almost but not entirely Australoid, and the women are, more frequently than the men, partly Mongoloid. The Asuras, hereditary blacksmiths and smelters, are somewhat Negroid as well, and Tasmanoid or Negroid individuals can be found marginally among the Munda. What we have here is a blend of at least three stocks—Negrito, Australoid, and Mongoloid, in varying proportions, with the Mongoloid partially linked to the female sex.

Moving up to the Khasi Hills we found a light-skinned and essentially Mongoloid people living above the mosquito belt of 4,000 feet. Linguistically and culturally they are tied to Burma and Indo-China, if not indeed to Polynesia. Their plateau is wet as well as chilly; it has the world's heaviest rainfall. Over the edges of the plateau, down the slope, other peoples are settled like layers in a cake; in the next set of hills, at 2,000 feet or so, live the Garos, much darker-skinned than the Khasis, and less frequently Mongoloid. Later, in Ceylon, we found the same thing. The Singhalese living on the steamy plain are long and lean in habitus, long headed, narrow faced; as one moves upward through Kandy to Nuwara Eliya, at 6,000 feet, they become shorter, stockier, and lighter skinned; the frequency of epicanthus also increases with altitude.[4] Here we have in two separate parts of the subcontinental area an identical cline linking Mongoloid racial characteristics with altitude, similar to that known to exist in Madagascar (for a theoretical study of racial clines, see Womble, '51: 315-322).

However, the world's sharpest racial frontier is probably the vast lip of the Himalayas along the whole southern border of Tibet and southwestern China, from Kashmir to Burma. All along the arc, above 14,000 feet, are Tibetans, fully Mongoloid, whether called Bhotias, Sherpas, or whatever. Below them come a host of tribes: Dotiyals, Magars, Gurungs, Tamangs, Rais, and Limbus in Nepal; Lepchas in Sikkim, unclassified in Bhutan; and over in the Northeast Frontier Area Mikirs, Miris, Abors, and Mishmis, and finally Nagas. These peoples are variously Mongoloid depending on altitude. Many of them, like the Lepchas and Limbus, actually look quite European in an Alpine sense, and in fact resemble the Caucasoid element in the Indonesian complex, particularly, in terms of the present report, the Formosan Atayals.

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[4] This will be shown in an extensive series of Singhalese measured by the late Dr. J. R. de la H. Marett, now being written up by Howard Stoudt of the Harvard School of Public Health.

That malaria may play a selective role in keeping the Mongoloids above 4,000 feet is no new idea to the Mongoloids themselves. They fear the disease and most of them know better than to try to live at a low altitude. However, something else must operate to keep the Caucasoids below 14,000 feet. That factor may well be a lack of high altitude adaptation concerned with libido and the oxygen transfer from mother to foetus; this is known by analogy from studies in Peru and Bolivia. Even among the altitude-adapted Tibetans, libido increases markedly on visits to an altitude of 4,000-7,000 feet. My authority for this is a man in a good position to know; the former police-chief of Kalimpong.

At the foot of the Himalayan slope stretches a vast forest, the Terai, filled with savage beasts. Although now nearly uninhabited, it was occupied within a century ago by a wild, jungly people with black skins and thick lips, who hunted in its shaded aisles with bows and arrows. Today some of the Munda-speaking peoples of Central India have moved up to its borders to work on tea plantations and the railroad.

Among the Aryans themselves, a distinction may be made between those who live on hilly country and those who cling to the flat Indo-Gangetic plain, like fish in an aquatic environment. Col. Clark suggested that the hill-people may represent an invasion of shepherds travelling on foot or horse, and the plainsmen one of cattle people riding in ox-carts; he is working on a linguistic basis for this distinction. Guha, too, pointed out that the distribution of Aryans in Assam coincides precisely with the altitude map, covering the flatland where bullock carts can go without road-building, and the moment one leaves the plain, he finds himself among more or less Mongoloid people speaking non-Aryan languages. In Nepal the Khass, the warrior caste of Aryans stretching from one side of the country to the other at about the elevation of Kathmandu, presumably represent the nonvehicular wing of the Aryan migration. The Newaris, the highly civilized Tibeto-Burman speaking occupants of the Kathmandu valley, who are part Hindu and part Buddhist, look for all the world like Chinese.

In Cochin we saw that cities as well as mountains can serve as refuge areas. There we walked down the famous street of the White Jews, who have lived there for nearly two millenia, keeping themselves out of the sun's direct rays under roofs and lattices, still as white as their ancestors in Palestine could have been; however, only 150 out of a former 1,500 are left. Thus through a choice of occupation and man-made environmental protection, bolstered with a breeding pattern of their own choosing and enforced by religious sanctions, men can produce their own isolates. Culture, in other words, is as much a part of our total environment as solar radiation and altitude, and culture itself may be a product of man's physical differentiation. The thing is circular.

After India even Paris is a letdown. On the rest of our trip we measured 7 Mahri-Socotran-speaking Arabs at Dammam, where Dr. Charles Matthews of Aramco was linguistically debriefing them. Thus I was able after over 20 years to make a correction for Bertram Thomas's erroneous head lengths in his still unique anthropometric series of these people (Thomas '32: Appendix I; Coon '39: 427-428). They are not, as his measurements implied, and pictures belied, microcephals.

In Rome we stayed with the hospitable Baron and Baroness Alberto C. Blanc, and saw his hand-axe site at Torre in Pietra; in Florence we stayed with Professor Lidio Cipriani, from whom I obtained a selection of 1,000 photographs of racial types for my projected *Races of the World.* I did a little business at the Musée de l'Homme in Paris and then reached home bewildered and exhausted. Only now are sights I saw and words I heard beginning to sort themselves out at all clearly, permitting me to write a report as old-fashioned and frankly preliminary as this one.

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THE STUDY OF NATURAL SELECTION IN PRIMITIVE AND CIVILIZED HUMAN POPULATIONS

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THE principle of natural selection as a guiding factor in human evolution is today universally accepted. However, although in recent years the mathematical papers of Fisher, Wright, Haldane, and others have greatly advanced our understanding of the manner in which the changes in gene frequency which constitute biological evolution come about, our knowledge of the actual workings of natural selection in human populations is almost nil, but little advanced today beyond that expressed by Wallace in 1871 in his well-known paper on "The action of natural selection on man." There are currently evidences, as in a number of recent essays, of a growing effort to extend our insight into many aspects of the problem (e. g., Chance and Mead, '53; Etkin, '54; Coon, '50, 55; Dobzhansky and Allen, '56). In a way, these essays as a group only point up the gap between the apparent response to selection which has characterized human evolution and our lack of detailed information with regard to the relation between natural selection and specific human characteristics. Field studies which have included as a stated objective the analysis of selective pressures in human populations, such as those of Birdsell ('50, '53) and Kluckhohn and Spuhler (Kluckhohn and Griffith, '50; Spuhler, '50), can almost be counted on the fingers of one hand. Contemporary man cannot be understood without a vast array of such information. Furthermore, at this moment one of the most actively discussed topics in human biology is the genetic risk of the increased amounts of ionizing radiation to which human populations all over the world are being subjected. A critical factor in these discussions is the nature of the assumptions made concerning the amount and manner of action of the selection to which today's man is subject.

When one contrasts the harshness of life for the African Bushman or the American Eskimo with conditions in the United States today, it would at first glance seem almost self-evident that we who enjoy the latter are functioning in a cotton-batting existence where each zygote, whatever its frailties, is—subject to a few accidents and socio-economic happenstances—virtually assured of a long, sheltered, and—more important from the genetic standpoint—almost equally reproductive life. This self-evident proposition has been repeated in recent years with an almost hypnotic regularity, with relatively few raising a doubting hand. Notable among these latter is S. J. Holmes, who wrote: "To talk . . . of natural selection as if it were practically done away with among civilized human beings, as many writers have done, is eminently absurd. In fact, it may be doubted if civilization has effected much diminution in the intensity with which natural selection acts on the human species. While much progress has been made in the art of saving life, there are many new forms of selective elimination which have been brought into play as a result of our civilization, and especially of our industrial development" ('24: 355).

Those who urge that the selective pressures on human populations are rapidly diminishing often cite the decrease in infant mortality and the preservation of persons with genetically determined diseases as proof of this thesis. It is true that infant mortality, rather than being the 500 per 1,000 of some primitive tribes, is now approximately 30 per 1,000 in the United States. This undoubtedly has significance from the standpoint of natural selection. But yet we are in no position to reach a judgment as to how much infant mortality is meaningful in the selective sense and how much is fortuitous, in the sense that any infant, regardless of genotype, who happens to be in a particular place at a particular time is destined to die. It is also true that modern medical practice is preserving the lives of many children and adults with genetically conditioned disease who a few centuries ago would have died within a short time after the appearance of their disease. And yet, to consider only a single example, juvenile diabetes, while undoubtedly a certain number of these children live and reproduce who would not have done so before the introduction of insulin therapy, reproductive expectancy as a whole is still greatly reduced. While the general principle that there has been some relaxation in certain types of natural selection seems sound, it is important to recognize that the genetic consequences of the apparent relaxation in these types of natural selection are not clear, nor is it any clearer where these apparent types of selection fit into the total picture of selection on human populations.

The general thesis which I would like to present is the following: The need is great for a variety of parallel studies on selective factors in advanced and primitive societies. Such studies are at the same level of importance as investigations on spontaneous mutation rates, the nature of the gene, skeletal remains, or the effects of irradiation on human populations, in our efforts to understand man's past and to predict his future. These studies, especially as they pertain to primitive groups, present real practical and methodological problems, and for these reasons, as well as a relative lack of interest, progress in this area has lagged far behind that in the other areas just mentioned. Yet by careful selection of projects, important advances can be made in the relatively near future. Finally, there is a certain urgency to the problem, so rapidly are primitive societies being disrupted.

This thesis is for the most part so broadly worded that it would appear to be the genetic equivalent of a political platform which is against sin and for mother love. However, there is one specific point on which considerable disagreement might arise, and this is with regard to the present feasibility of significant studies on natural selection. It is to this general question that the remainder of this presentation will be devoted. More specifically, three different areas will be examined in which the time would seem to be at hand for some rather significant observations. Dr. Levine has discussed still a fourth area in this same symposium. It should perhaps be emphasized that these areas are arbitrarily selected as examples, although, as will become apparent, there is a certain design in the choice of these examples.

The Variability in Individual Reproductive Performance in Primitive and Civilized Cultures

In the strictly biological sense, the genetic worth of a newly formed zygote is measured in terms of the number of descendants which will survive it. In the broadest sense, selection is operative when in a population the average reproductive performance departs from simple replacement values and/or individual members of the population contribute disproportionately to the next generation. It is this latter aspect of selection, between individuals of the same species, which has attracted the most attention and is usually thought of when the word "selection" is used. This selection may be either on biological or cultural grounds. It will often be difficult to distinguish between the two types. The focal point of this essay is to define representative areas in which the *changes* in selective pressures inherent in the transition from a relatively primitive to a relatively advanced culture can be isolated and characterized. Studies of the manner in which communities and the individuals of whom they are composed replace themselves offer unusual opportunities from the comparative standpoint. The differences in individual fertilities in contemporary Western civilization and their possible genetic implications have been the subject of repeated comments. No one seems to have improved materially on Pearson's statement of 60 years ago: "It is a point which seems to me of the utmost significance that, allowing for the proportion of unmarried in the population, about ⅕ to ⅙ only of the adults produce quite one-half of the next generation, and any correlation between inheritable (physical or social) characteristics and fertility must thus sensibly influence that next generation" (1897: 83). Very much less has been written concerning fertility differences in primitive societies. Reliable data on this subject are scarce, since the early explorers and even anthropologists were little concerned with studying the fertility of the natives, and by the time the interest of the problem was recognized, the primitive state was seriously disturbed. Much of what is available has been compiled by Krzywicki ('34). As he points out, it is unsatisfactory other than as a very rough guide. Thus far, despite a considerable search of the literature, no single description of the distribution of individual completed fertilities in a truly primitive "hunting and gathering" society has come to light.

However, in recent years, extensive and fairly reliable data have become available for a number of relatively unsophisticated areas. These are all beyond the hunting and gathering stage and well into the agrarian epoch. By way of examples, table 1 gives some recent data from Africa and Pakistan. The sociological factors which may have altered the findings from the situation which existed several hundred years ago are discussed in detail in the original presentations. In addition, the figures are subject to a number of errors, also discussed in detail in the original presentations. Furthermore, to be entered into the table, a woman must have survived the childbearing period. The three sets of data are not strictly comparable, but agree on the important point of the wide distribution of individual fertilities.

TABLE 1

Number of children ever born to women aged 45 years and over, in four different areas

Locality Number of children ever born	Agogo Township, Ashanti Territory The Gold Coast *	Two villages East Pakistan †	Liberia ‡	New South Wales, Australia §	U. S. A.**
0	11	2	7	1,110	2,622,060
1	8	2	4	583	1,459,460
2	19	3	7	581	1,676,700
3	14	6	1	644	1,318,940
4	29	3	4	702	962,780
5	26	9	6	813	1,179,400
6	34	10	5	855	
7	39	15	8	976	787,200
8	31	5	7	963	
9	26	8	1	847	
10	32	5	3	786	331,220
11	16	5	1	568	
12	6	4	1	422	
13	2	1	2	226	
14	1	1	1	129	
15	1	0	1	57	
16	0	0	0	39	
17	1	1	0	12	
18	0	0	0	5	
19	0	0	0	2	
20	0	0	0	2	
21	0	0	0	1	
22	0	0	0	1	
23	0	0	0	0	
24	0	0	0	1	
30	0	0	0	1	
Total	296	80	59	10,276	10,337,760

* Collected during one fortnightly (field study), 1945. v. Fortes 1954.

† Collected over several months (field study), 1953. v. Aird 1956.

‡ Collected over several months, 1956. I am most grateful to Drs. A. H. Jonkers and Frank Livingstone for permission to use these unpublished data, based on the reports of post-menopausal women living in the native villages of the Firestone Plantation.

§ Collected in 1898-1902, Powys, 1905. Unlike the other three studies, this concerns women aged 46 and over.

** U. S. Decennial Census, 1940. Government Printing Office. The entry "single women" in the census data has been equated to childless.

There is evidence from many parts of Africa that at present infertility is more common than was the case 20 or 30 years ago. This is often attributed to the spread of venereal disease, but of course the introduction of venereal disease to Africa goes back much further than that. It is not at all clear to what extent the figures given in table 1 depart, for this and other reasons, from those which might have been observed several hundred years ago. Data from other comparable areas in Africa show about the same amount of infertility (*e. g.*, Mitchell, '53), and it may be that even under circumstances most favorable to reproduction, a minimum of 3 to 5% of women will remain childless. Under unfavorable primitive conditions, the sterility rate may be appreciably higher. Thus, Malaurie, Tabah, and Sutter ('52) record that among 51 postmenopausal Greenland Eskimos, 8 were sterile.

How do these figures compare with those for contemporary cultures? Fisher ('30)—and before him, Pearson—has argued that the most important cause of genetic change in Western civilization is differential fertility. In his argument, Fisher drew heavly on the data of Powys ('05) on the number of children ever born to 10,276 married women dying age 46 or over in New South Wales from 1898-1902. These data are also reproduced in table 1. There is room for debate as to how typical data from Australia at the turn of the century are for Western civilization in general, but unfortunately, such is the paucity of good data on this problem, these were apparently the best Fisher could find. In these data, the most fertile 50% of the women gave birth to 76.4% of the children. By comparison, in Agogo 50% of the women were responsible for 69.7% of children ever born, while for Pakistan and Liberia the corresponding figures are 69% and 78.5% respectively. It would appear from these data that differences in individual fertilities, of a magnitude which led Fisher to consider differential fertility as the most important agent of genetic change in contemporary society, probably extend far back in the history of man.

Data on familial correlations in fertility are as inadequate as data on the distribution of individual fertilities. Fisher ('30) in an analysis of Pearson's (1899) data concerning the numbers of children born to mothers and daughters in a thousand cases extracted from the British peerage, finds a correlation of 0.2096 for the number of children born to mothers and daughters, and a correlation of 0.1123 for the same relationship with respect to granddaughters and their paternal grandmothers, and develops reasons for regarding this in some indeterminate amount as genetically determined.[1] He then writes: "We have seen that by no means extreme genotypes have rates of reproduction in a ratio higher than 2 to 1, and if for simplicity we imagine a population consisting of two strains, which, having equal mortality, differ only to this extent in fertility, it is easy to see that the numbers of one type relative to that of the other will, in ten generations, be increased over a thousandfold. To put the matter in another light, if at the beginning of the period the population consisted of 97% of the less fertile and 3% of the more fertile strain, in 5 generations the 2 strains are brought to an equality, and in 5 more their situations are reversed, so that the less fertile strain is represented by 3% and the more fertile by 97% of the population. Civilized man, in fact, judging by the fertility statistics of our own time, is apparently subjected to a selective process of an intensity approaching a hundredfold the intensities we can expect to find among wild animals, with the possible exception of groups which have suffered a recent and profound change in their environment. We may therefore anticipate that a correspondingly rapid evolution has taken place within historical times in the appropriate mental attributes" ('30: 199).

The developments as regards differences in individual fertilities since Fisher wrote these lines would apparently only serve to reinforce his argument. Thus, table 1 gives data concerning the number of children in completed families in the U. S. in 1940, as revealed by the census carried out that year; the condensed form in which the data are presented hinders certain exact calculations, but by the time of the 1940 census, 50% of the women had become responsible for approximately 88% of the children born each generation.

In our present monogamous and relatively long-lived society, specifying the reproductive performance of the female of the species tends to describe the distribution of male fertilities as well. But in societies where there is polygamy and marked social pressure favoring marriage, this would not be the case. In a stimulating essay entitled "Does the fittest necessarily survive?" Shubik ('54) works out the elementary mathematics of survival in a society where the "strong" regard each other as a threat and so are thrown into conflict, while the "weak" are ignored as presenting no challenge. But to the extent that in a society with very high marriage rates the weak later inherit, if not the earth, at least some of the widows of the strong, not only do the fittest not necessarily survive, but the reproductive differential would be even more in favor of the weak than the mere facts of life expectancy would suggest.

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[1] Correlations regarding fertility must be regarded in a different light than, for example, correlations regarding stature in successive generations. For example, a short person who fails to reproduce will be dropped from further consideration regarding correlation in stature between various generations, but may appear in certain fertility statistics, depending on how one elects to treat the data. This introduces complications into how one views and contrasts fertility correlations with other biological correlations.

Two obvious conclusions emerge from these figures and considerations: (1) Striking differences in individual fertilities are not new but, with all their important genetic consequences, probably extend far back into the history of the human race. (2) These differences, considered in terms of individuals reaching reproductive maturity, are probably more pronounced today than ever before, although, in terms of reproductive expectation *at birth*, the change may actually be in the opposite direction. These are facts with which we would all agree. There can be no doubt of the evolutionary implications of these differences. Their implications from the standpoint of natural selection depend on the degree to which they are genetically determined. If there were no genetic component in fertility differences, then, while they might be a mechanism for human evolution, it would be independent of biological selection. From our present knowledge of inherited defects which directly or indirectly interfere with human reproduction, there can be no doubt that fertility is to some extent genetically determined, and that natural selection is therefore to some extent involved in these fertility differentials. Specification of the magnitude of this component depends not on further studies of the British peerage or German nobility (cf. Editorial Note, 1935), but on detailed studies of relatively stable populations not subjected to rapidly evolving socioeconomic circumstances.

At the one extreme it can be argued that if the degree of differential fertility which has obtained over past centuries has facilitated man's evolution, then the present increase will aid the adjustment of the human species to rapidly changing conditions. At the other extreme it may be argued, as Fisher ('30) so compellingly has (see also Pearson, 1897), that our present civilization replaces an earlier, eugenic set of fertility determinants with an essentially dysgenic set. The truth may not be found in either extreme. More specifically, it seems likely that some of the presumably eugenic facets of differences in individual fertilities as seen in primitive societies are still with us, but that the dysgenic facets are now more numerous than they were previously. Whether the net balance is still eugenic, in permitting appropriate genetic responses to a rapidly changing environment, even if more slowly, is a question of the utmost consequence. But whatever one's interpretation of the cause or the precise genetic consequences of the differentials, it is clear that because of these differentials the potentiality of change in the composition of the gene pool, which *is* biological evolution, is today enormous. It follows that knowledge of fertility determinants is of prime importance.

Much has been written concerning cultural determinants of fertility, as reflected in differences in mean reproductive performance between various socioeconomic groups. But within any group, there are still great ranges in fertility. To what extent these individual differences are cultural, and to what extent biological, is difficult to say. For instance, is the overwhelmed couple of today which limits its family to one child because it cannot plan for more, the modern counterpart of the Australian aborigine and his mate who killed all their children but one because they interfered with their mobility?

A comprehensive understanding of the occurrence and the cause of individual fertility differences in various societies is certainly one of the outstanding genetic objectives of our day. The timeliness of data on the very primitive societies needs no emphasis. That each society will have to be evaluated as a separate problem is obvious. Consider, for example, the matter of differences in marriage rates between societies. In the 1940 census data some 11.5% of the women were unmarried, which, in our culture, generally excludes reproduction. On the other hand, in the Ashanti village which formed the basis of the calculations given earlier, very few individuals other than the grossly defective remain unmarried or widowed (Fortes, '54), a situation characteristic of all Africa (cf. Martin, '53; Roberts, '56). In the Pakistan village studied by Aird ('57), the marriage rate was 100%; such high rates were apparently typical of almost all primitive societies (Krzywicki, '34). Now, it is common to think of a high rate of celibacy as dysgenic. In the case of women in the United States, there is an inverse correlation between years of education and marriage rate. To the extent that the more educated women tend to be a more intelligent group, their failure to marry means the loss of desirable genes from the gene pool. However, a marriage rate of 100% also has certain dysgenic implications. Two illustrations will suffice. In multiple neurofibromatosis, the fertility of affected individuals in the state of Michigan at the present time is 0.527 that of their normal siblings, with females manifesting a higher fertility than males (Crowe, Schull and Neel, '56). However, only 32% of affected males marry, in contrast to 58% of affected females. As shown in table 2, the fertility of married affected individuals is 0.795, with females again more fertile than males. A proportion of those who fail to marry are so severely handicapped by their disease that it is doubtful whether they would marry in any society. However, another proportion fails to marry, not for reasons of physical disability but because of the disfiguring effects of the disease or its effects on intelligence. It seems likely that in a more primitive society these latter individuals would marry and propagate their defect. As a second illustration of the dysgenic implications of a marriage rate of near 100%, we might consider the relation between I. Q. and fertility. Penrose ('50) has argued that since both the very intelligent and the very unintelligent show relatively decreased rates of reproduction, there may exist a balanced polymorphism with respect to the genetic determinants of intelligence. But if, as seems possible, the intelligence requirements for marriage are being pushed upwards, then the net effect of this trend would be to shift the fulcrum of this balance toward the upper end of the scale. Whether this would offset the effects of the lower marriage rates of the more educated is debatable. Thus, in considering the genetic implications of failure to marry, it seems likely that each society will require a separate balance sheet.

TABLE 2

Fertility in neurofibromatosis, after Crowe, Schull and Neel, 1956 *

Fertility all patients with neurofibromatosis	0.527
Fertility male patients with neurofibromatosis	0.413
Fertility female patients with neurofibromatosis	0.748
Fertility all married patients with neurofibromatosis	0.795
Fertility male married patients with neurofibromatosis	0.620
Fertility female married patients with neurofibromatosis	0.887

* Fertilities are expressed in the fraction of the fertility of unaffected siblings.

As important as the causes of differences in individual fertilities, is the problem of differences in the survival rates of the children of various families, especially in primitive cultures. A related problem is the degree to which pregnancy wastage and infant and childhood mortality is disproportionately greater in large than in small families.

Krzywicki emphasizes the fact that infanticide has apparently been widely practiced by primitive societies as a means of reconciling population with available resources. If in primitive societies there were strong social pressures for modest-sized families, say two or three, then here is a factor to offset differential fertility; a woman who had completed ten pregnancies might, because of infanticide, rear no more children than a woman completing four pregnancies. It is interesting to contemplate the possibility that if this were the case, then by a very indirect means we may be returning to the same state of affairs. Family planning in this country increasingly converges on two or three children as an optimum (Freedman, Goldberg and Sharp, '55). With the wider dissemination of birth control information and education, the striking differentials in individual fertility shown in table 1 may tend to diminish appreciably in the near future. Indeed, there is evidence that this process has already begun.

The role of genetic drift in human evolution has been the subject of repeated speculation. The possible significance of drift in a population is inversely proportional to the effective breeding size of the population. Effective breeding size (N_e) was defined by Wright ('38) in terms of the number of parents in the population (N), and the variance in the average number of children (gametes) by which each parent is represented in the breeding population of the next generation (σ_k^2). To an approximation,[2]

$$N_e = \frac{4N - 2}{\sigma_k^2 + 2}.$$

In a sense, it is the significance of σ_k^2 which we have just been discussing. Since this term appears in the denominator of the expression which defines population size, factors decreasing its magnitude will increase effective population size. The trend in Western culture toward planned families of two to 4 children (cf. Freedman, Goldberg and Sharp, '55) thus has important implications for the concept of effective breeding population. Equally important is the increasing range of mate selection possible to the average individual, *i. e.*, the increase in N, which appears in the numerator of the expression defining effective population size. There are, then, two different factors at work today to increase effective population size and so to minimize the role of drift in human evolution. Drift which leads to the chance elimination of favorable genes is of course dysgenic. The obvious implication is that future populations will be more protected than primitive ones against one undesirable type of genetic change.

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[2] This approximation holds only when the breeding size of the group remains the same in successive generations. However, departures from this condition only introduce another term in the expression.

The effective breeding size of a human population is in actual practice difficult to define. This is well illustrated for a primitive population by the excellent study of Roberts ('56) on a Dinka village of the southern Sudan, and the equally excellent study of Spuhler and Clark (unpublished) on the somewhat less primitive population of Ann Arbor, Michigan. These difficulties notwithstanding, it is clear that the effective size of many primitive populations was small indeed, with the size subject to still further sudden and sharp decreases periodically because of the inroads of war, pestilence, and famine. Indeed, one who pursues these considerations is sometimes left wondering why the minor geographical subdivisions of the human race are not more strikingly different than they are. The answer perhaps lies in the apparently ceaseless migrations (and intermixtures) and custom-sanctioned exogamy of human populations.

Treatments of the dysgenic effects of ionizing radiation have thus far not taken into account the implications of the wide differences in individual fertilities. Two such implications in particular deserve mention. First, the probability of loss from the gene pool, which is already high for most recently mutated genes, will become even higher with such individual fertility differences as exist in human populations; on the other hand, the surviving newly mutated genes may undergo a relatively rapid increase in numbers, with a corresponding increase in the probability of homozygosity with respect to the mutant involved. For mutants with serious deleterious effects, the consequence would be an increased rate of elimination, with an earlier return to the equilibrium state than would be the case if each irradiated individual had the same chance of propagation. This consequence would appear to be of greater relative importance in small than in large populations. Second, to the extent that fertility is a nonspecific indicator of physical vigor, and to the extent that most nominal recessive mutants have slight heterozygote effects, it seems possible that in an irradiated population individuals who either by chance or by virtue of their radiation experience had accumulated significantly more mutant genes than the average might suffer from impaired fertility, and so fail to transmit their mutant genes to the next generation.

The Genetic Significance of Changing Dietary Patterns

The second area to be discussed as illustrative of the opportunities for the study of natural selection in human populations is concerned with changing dietary patterns and their relation to the pattern of morbidity and mortality in a population. We shall be forced to limit our considerations to a single cause of death, heart disease, the leading cause of death in the United States today. Figure 1 shows the age at death from arteriosclerotic, hypertensive, or " degenerative " heart disease in the state of Michigan for 1953. Some 3.7% of these deaths occur prior to age 44, and another 3.7% between the ages of 45 and 49. Moreover, hypertension, the predisposing factor in hypertensive heart disease, may result in relatively early death because of cerebral hemorrhage

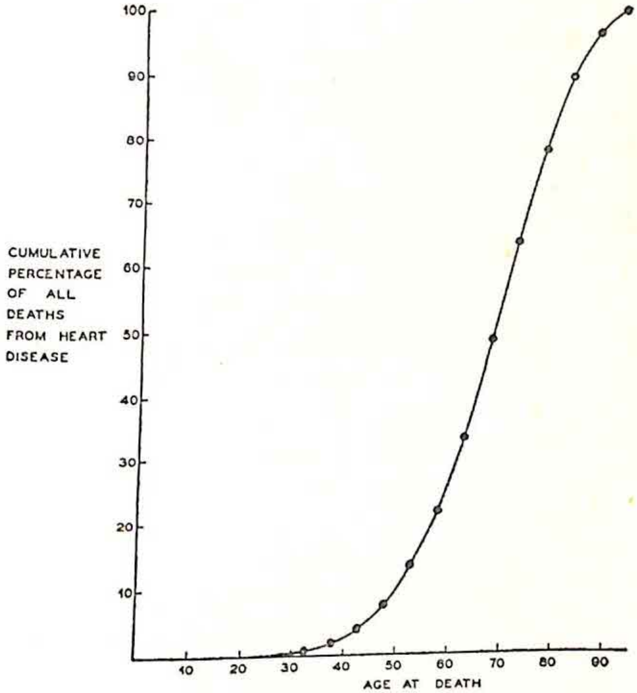


FIG. 1. CUMULATIVE FREQUENCY OF AGE AT DEATH FROM ARTERIOSCLEROTIC HEART DISEASE, NON-RHEUMATIC CHRONIC ENDOCARDITIS AND OTHER MYOCARDIAL DEGENERATION, AND HYPERTENSION WITH HEART DISEASE, IN THE STATE OF MICHIGAN FOR 1953. THE FIGURE IS BASED ON A TOTAL OF 20,546 DEATHS; THESE CONSTITUTED 33.1% OF ALL DEATHS IN THE STATE FOR THAT YEAR.

or renal failure. In contrast, at least one of these important causes of severe heart disease, essential hypertension, is apparently much less common in many other cultures than in our own (review of literature in Bays and Scrimshaw, '53), and there is reason to suspect that one other cause of death from heart disease, arteriosclerosis (and its predecessor, atherosclerosis), may also be less common. The reasons for this have been extensively discussed; Murrill ('55) in particular has brought out the difficulty, on the basis of the existing data, of reaching conclusions concerning the relative importance of intrinsic (genetic) and extrinsic (environmental) factors in such differences as have been reported between various racial groups. In the following discussion, we shall not be concerned primarily with the question of genetic differences between racial groups in predisposition to various types of heart disease, but with the implications of changing environmental conditions on the expression and evolution of a given genotype. We shall limit our attention to two facets of the problem, namely, the effects of changes in dietary fat and salt intake.

Let us consider first the matter of dietary fat. By and large, in the world today the average intake of animal fat is proportional to the standard of living. Since the standard of living is rising, so also is the animal fat (and total caloric) intake; the relation of this to the apparent increase in coronary artery disease has provoked an extensive and controversial literature, which we cannot review exhaustively. As of this writing, the following statements seem justified: Individuals developing so-called coronary heart disease, including those in whom it appears at a relatively early age, tend to have somewhat elevated blood cholesterol levels, as well as altered phospholipid-cholesterol ratios (Davis, Stern and Lesnick, '37; Poindexter and Bruger, '38; Lerman and White, '46; Collen, '49; Adlersberg, Parets and Boas, '49; Gertler and Garn, '50; Gertler, Garn and Lerman, '50; Morrison, '52). These levels and ratios can be correlated with animal fat intake and altered by dietary measures (Dock, '50; Keys, Mickelsen, Miller and Chapman, '50; Hildreth, Mellinkoff, Blair and Hildreth, '51; Priddle, '51; Allen, Katz, Keys and Gofman, '52; Byers, Friedman and Rosenman, '52; Keys, '53; Pomeranze, '53; Keys and Keys, '54; Keys, Vivanco, Rodriguez Minon, Keys and Castro Mendoza, '54; Bronte-Stewart, Keys and Brock, '55; Keys, Anderson, Fidanza, Keys and Swahn, '55; Beveridge, Connell and Mayer, '56; Bronte-Stewart, Antonis, Eales and Brock, '56; Nelson, '56). There is evidence that serum cholesterol levels in individuals on the "average"

American or European diet are to a certain extent under genetic control (Wilkinson, Hand and Fliegelman, '48; Stecher and Hersh, '49; Adlersberg, '51; Schaefer, Drachman, Steinberg and Adlersberg, '53; Piper and Orrild, '56), although, for the milder degrees of hypercholesterolemia, the role of familial dietary similarities has not been properly investigated. It may thus be argued that diets relatively rich in animal fats (and generous in total calories) bring to light genetically determined differences in the ability to handle fat, differences which then become subject to natural selection. An additional (indirect) factor in bringing to light these differences may be the increasingly sedentary life of Americans, since a group of young individuals on a high fat, high calorie diet showed no elevation of serum lipoprotein and cholesterol levels as long as they exercised sufficiently vigorously to prevent weight gain, but did show such elevations in the absence of exercise and with weight gain (Mann, Teel, Hayes, McNally and Bruno, '55); but see Keys, Anderson and Aresu, '56). Thus, the greater physical activity of primitive man may have tended to keep his blood lipid levels low despite a diet relatively high in animal fat; this would help explain why selection has not previously operated on this system to a greater extent.

Most of the cholesterol in the blood is associated in complexes with proteins and lipids; there is a considerable range in the size of these molecular complexes. In persons with coronary heart disease there is a tendency for these molecular complexes to be larger than in persons without coronary disease (Gofman, Lindgren, Elliot, Mantz, Hewitt, Strisower, Herring and Lyon, '50; Gofman, Jones, Lindgren, Lyon, Elliott and Strisower, '50; Gofman, Glazier, Tamplin, Strisower and DeLalla, '54; Lyon, Jones, Graham, Gofman, Lindgren and Yankley, '52). These molecular complexes are also responsive to dietary measures such as restriction of animal fat (Lyon, Yankley, Gofman and Strisower, '56). Whether determinations of serum cholesterol levels or studies of lipoprotein molecule size have a greater predictive value as regards susceptibility to coronary heart disease is not yet clear (Committee on Lipoproteins and Atherosclerosis, 1956).

Genetic studies on the control of serum lipoprotein molecule size have yet to be carried out. However, it is difficult to avoid the speculation that increasing levels of animal fat consumption as well as an increase in total caloric intake, coupled with decreasing physical activity, have rendered important an agent of natural selection which formerly affected only a "favored" few. To the extent that there is a genetic determinant in the ability to handle relatively high dietary fat loads without developing atherosclerosis, then biological natural selection should be at work in a society such as ours where one-fifth of the population over age 30, or about 15,000,000, are overweight, with an estimated 5,000,000 of these 20% or more above the ideal (Armstrong, Dublin, Wheatley, and Marks, '51).

You will have noted the emphasis on animal fat. This differs from vegetable fat chiefly in the greater degree of saturation or hydrogenation of the former. There is a growing body of evidence to indicate that the level of intake of vegetable fat has, on an equicaloric basis with animal fat, less tendency to elevate either serum cholesterol levels or the proportion of lipoprotein "macromolecules" (Kinsell, Michaels, DeWind, Partridge and Boling, '53; Ahrens, Blakenhorn and Tsaltas, '54; Beveridge, *et al.*, '56; Bronte-Stewart, *et al.*, '56). This statement holds true only for vegetable fats which have not been artificially hydrogenated, a practice now widespread because it renders some fats commercially more attractive. Hydrogenation, in increasing the degree of saturation of vegetable fats, reduces their content of the three so-called essential (unsaturated) fatty acids; these may play a role in the body's economy beyond that indicated by their degree of unsaturation alone (references in Rathmann, '57).

Let us turn now to the matter of salt intake. Until relatively recent times, salt was a rather precious commodity, with commerce in it often under government control. Today the salt consumption of the average individual in this country is probably 5 to 10 times greater than that of his counterpart in most groups of primitive man (Dock, '56). The role of this in the etiology of hypertension and hypertensive heart disease is debatable. However, it is a fact that rats placed on a level of salt intake equivalent to that of the average human diet will often develop hypertension (Meneely, Tucker, Darby and Auerbach, '53). It has also been reported that among 1,346 employees of the Brookhaven National Laboratory, the incidence of essential hypertension was significantly related to salt intake (Dahl and Love, '54, '56). There exist no data as to whether the ability to handle salt loads is at least in part genetically determined and hence susceptible to selection, but it would be surprising to me if it were not.

If, as mentioned earlier, heart disease of the types under discussion is uncommon in societies on a low-fat, low-salt diet, it will be interesting to observe the effects of a change in dietary habits on the morbidity picture in these societies. For instance, there is some evidence that among primitive Africans blood pressures tend to be low, whereas among the American Negro hypertension is more common than it is in Caucasians (references in Bays and Scrimshaw, ’53). Furthermore, it appears that certain categories of foreign-born students, coming to this country with relatively low blood pressures, over a ten-year period acquire blood pressures comparable with those of native-born Americans (Szent-Györgyi, ’56). What is the role of altered salt intake in this difference? How does this relate to concomitant changes in the pattern of living?

Heart disease is but one of a number of disabling and potentially fatal diseases encountered in this country among young adults. For example, there are the so-called collagen diseases. Over the world, primitive peoples are being projected in a few generations from a Stone Age to an Atomic Age culture. In making this transition, they will be called upon to telescope into a few generations biological and cultural adaptations which have extended over a period of thousands of years in Europe. To the extent that these adaptations involve genetic systems, here is a priceless opportunity to study biological selection. One of the most exciting opportunities today is that of collaborative studies along these lines between anthropologists, geneticists, and physicians all over the world.

The Abnormal Human Hemoglobins

The third area to be discussed as illustrative of the opportunities for the study of natural selection in man, concerns the abnormal human hemoglobins. Our first example involved a situation where the genetic consequences are reasonably apparent, but the responsible genetic factors, if any, are quite obscure. In the second example, both the genetics of the situation and its consequences were somewhat better defined. We come now to what is probably the clearest opportunity existing today to analyze in detail a particular genetic system subject to a known selective pressure, and the potential consequences of a change in that selective pressure.

Even if time permitted, the number of reviews of the human hemoglobins which have appeared in recent years render any extended consideration of the basic facts unnecessary (White and Beaven, ’54; Singer, ’55; Itano, ’55; Chernoff, ’55; Itano, ’56; Itano, Bergren and Sturgeon, ’56; Zuelzer, Neel and Robinson, ’56; Gatto, ’56; Neel, ’56, ’57). The reader is no doubt aware that the sickling phenomenon is due to a gene which when heterozygous produces a clinically asymptomatic condition known as the sickle cell trait, and when homozygous, the severe sickle cell anemia. In the sickle cell trait, 30 to 40% of the hemoglobin is of an abnormal type, known as hemoglobin S, whereas in the anemia, all the hemoglobin is of the abnormal type save for a fraction which may be of the fetal type. A second abnormal hemoglobin also encountered in Negroes is known as hemoglobin C. Genetically, it behaves in the same way as hemoglobin S, but the disease state due to homozygosity for the gene is much less severe than in sickle cell anemia. The genes responsible for these two abnormal hemoglobins appear to be either alleles or linked. The known distribution and frequencies of these two genes in West Africa, in which we are particularly interested here, are shown in figures 2 and 3. These relatively high frequencies are probably maintained primarily through the mechanism of a balanced polymorphism.

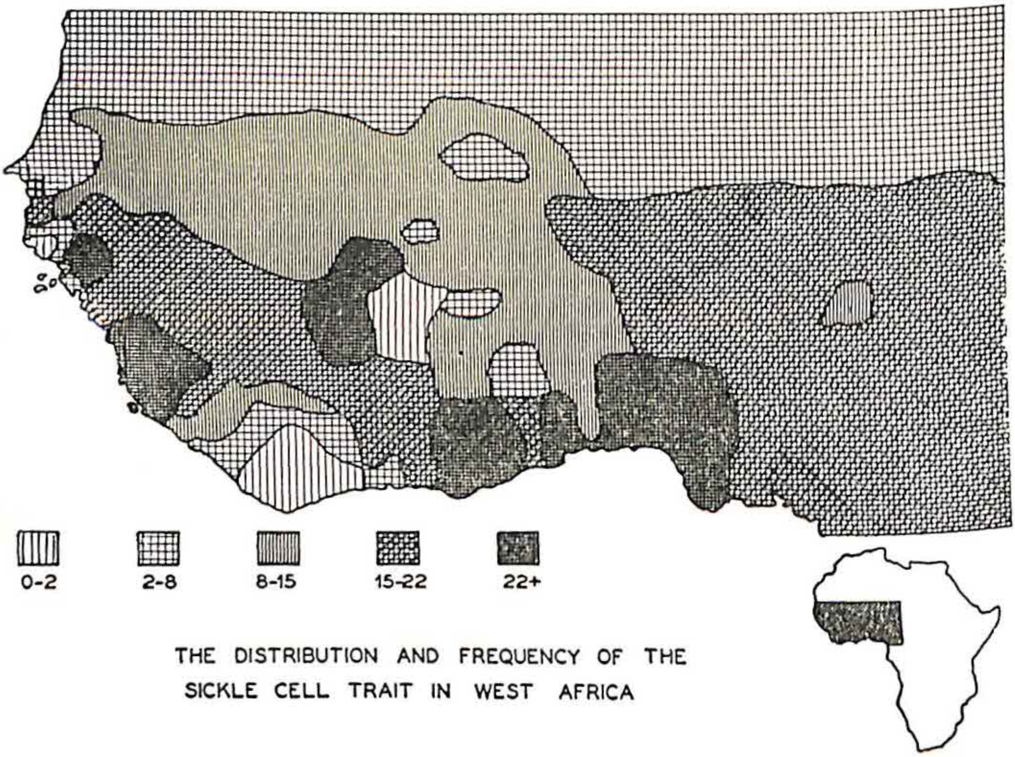

0-2
2-8
8-15
15-22
22+
THE DISTRIBUTION AND FREQUENCY OF THE SICKLE CELL TRAIT IN WEST AFRICA

FIG. 2. THE DISTRIBUTION AND FREQUENCY IN WEST AFRICA OF THE SICKLE CELL TRAIT, MODIFIED FROM LIVINGSTONE, 1957. THE MAXIMUM FREQUENCY UNDER THE ENTRY "22 +" IS 31%.

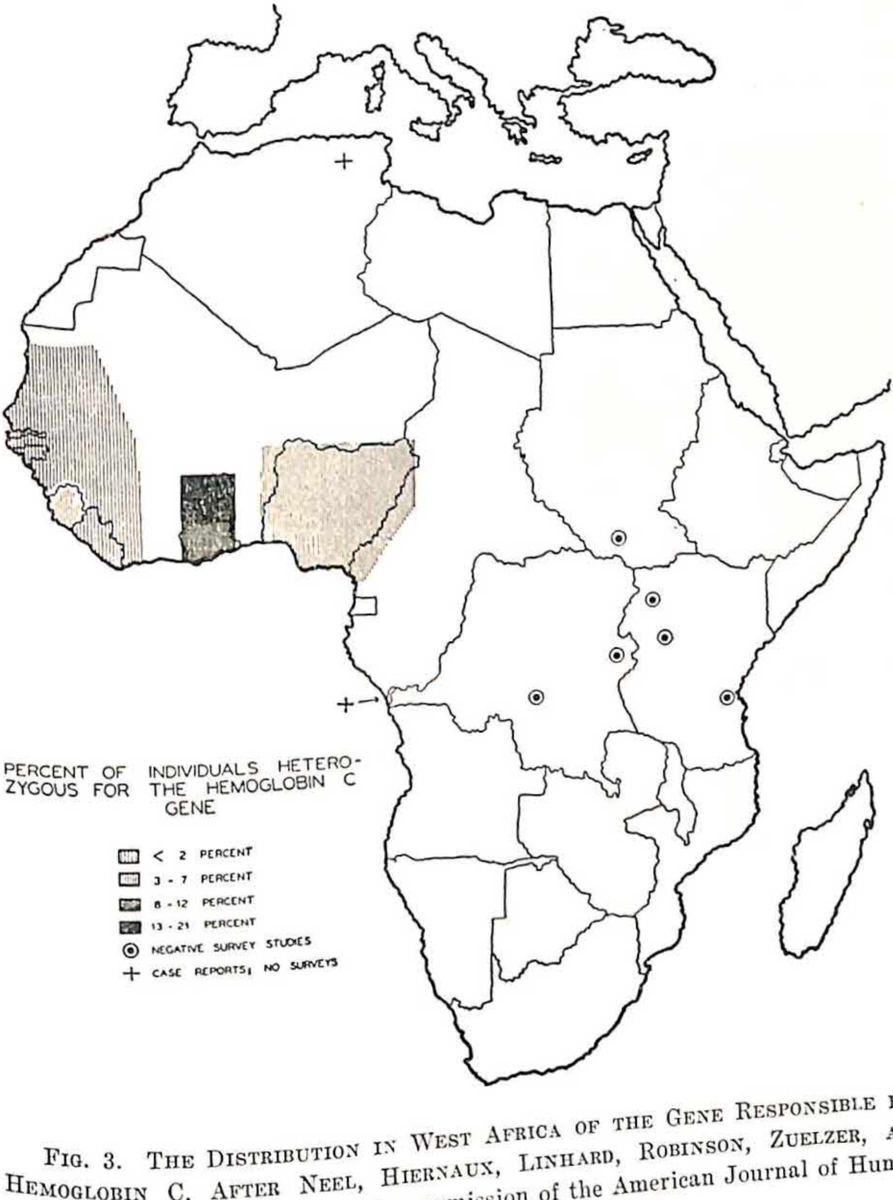
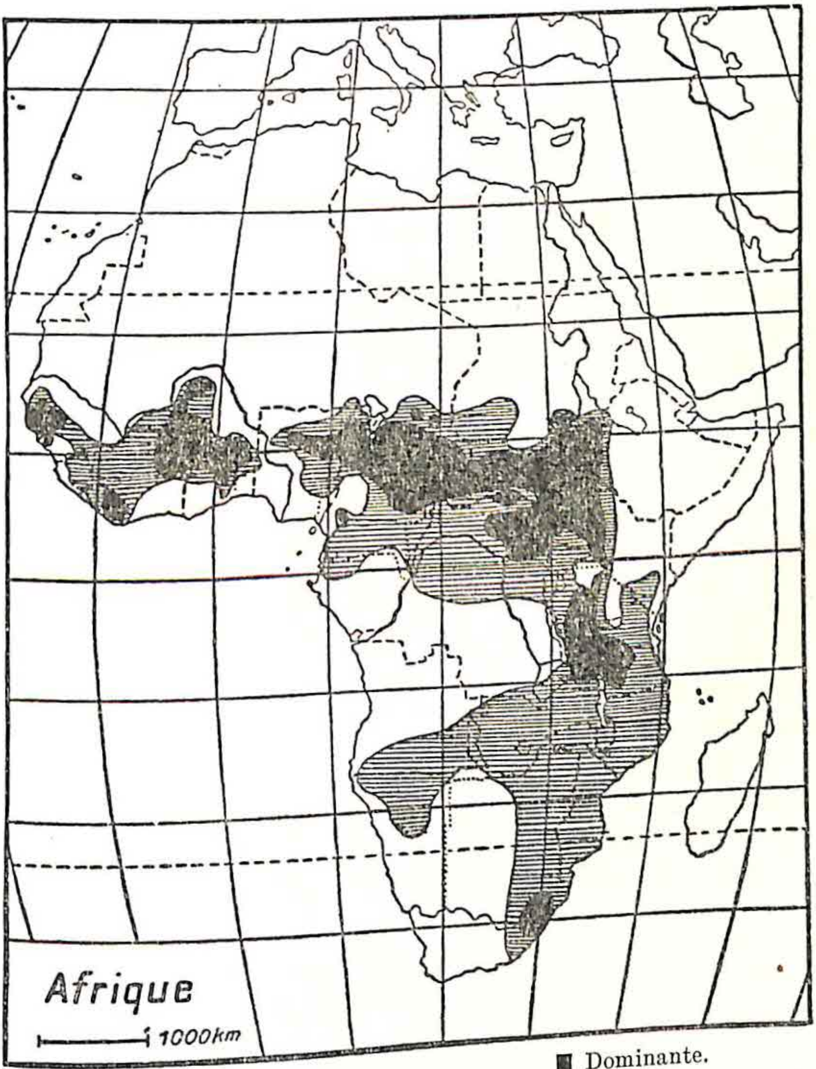


Fig. 3. The Distribution in West Africa of the Gene Responsible for Hemoglobin C, After Neel, Hiernaux, Linhard, Robinson, Zuelzer, and Livingstone, 1956 (reproduced by permission of the American Journal of Human Genetics).

The most plausible cause of this polymorphism yet advanced with respect to hemoglobin S is a lessened susceptibility of the individual with the sickle cell trait to the ill-effects of infection by *Plasmodium falciparum*. This thesis, so ably argued by Allison ('55), is undoubtedly familiar to the reader, but it seems unlikely that this constitutes the whole story. The mechanism responsible for the relatively high frequency of the hemoglobin C gene is unknown. Although the first possibility to be explored is again a differential susceptibility to malaria, data on this point have not yet been published.

All these facts are well known. There are some recent advances in our knowledge of the abnormal hemoglobins in West Africa which in my opinion open a new chapter in the study of these genes. There has been considerable speculation over the question of whether the sickle cell gene originated from one focus or multiple foci. This question may never be answered with finality. However, recent evidence makes it quite probable that in the western portion of West Africa at least, gene flow through migration supplies the major explanation of the distribution of the sickle cell gene, although the possibility of secondary, minor centers resulting from mutation and subsequent selection cannot be excluded with certainty. Our group has recently published data indicating a low frequency of the genes responsible for hemoglobins S and C among the tribes occupying the rain forests of southeastern Liberia (Neel, Hiernaux, Linhard, Robinson, Zuelzer and Livingstone, '56). Leite and Ré ('55) have also reported a low frequency of the sickling phenomenon in certain of the tribes found in Portuguese Guinea. Figure 4 indicates one interpretation of the areas of distribution in West Africa of tribes exhibiting a sufficiently distinctive and primitive culture to be regarded as more directly related to the early inhabitants of West Africa than many of the tribes now residing in that region (Baumann and Westermann, '48). It will be noted that of the three such areas in West Africa, two involve Portuguese Guinea and Liberia. On the basis of these facts, Leite and Ré have urged that the sickle cell gene is a relatively recent introduction into western West Africa, a point of view with which I agree. Otherwise stated, while the possibility of the origin of the sickle cell trait through mutation in West Africa cannot be excluded, there is evidence for a very substantial influx of the gene in consequence of relatively recent migratory flow originating east of the Gold Coast.

If we accept the point of view that the sickle cell gene owes its presence in western West Africa in whole or in part to an introduction



La civilisation paléonégritique.

▨ Dominante.
≣ Éléments importants.

FIG. 4. THE DISTRIBUTION OF "PALEONEGROID" ELEMENTS IN AFRICA. This figure is reproduced by kind permission from "Les Peuples et les Civilisations de l'Afrique," by H. Baumann and D. Westermann, published by Payot, Paris, 1948.

in the not-too-distant past, then some very obvious questions at once arise regarding the distribution of hemoglobin C. The "diffusion gradient" type of frequency distribution shown by this gene implies a relatively recent origin, with dissemination from a single focus. Is this origin pre- or post-migrational with respect to the sickle cell gene? If the origin is post-migrational, and if the time of the migration can be even approximately established, there is an unparalleled opportunity to document the date of appearance and subsequent spread of a "new" human gene with the resulting inferences about its selective value, inferences which can then be checked by direct observation.

There would seem to be two chief approaches to the question of the time of origin of the gene responsible for hemoglobin C. On the one hand, some information should come from a detailed study of the distribution of the gene in West Africa. As pointed out elsewhere (Neel, *et al.*, '56), if the origin of the gene is essentially pre-migrational, then one might expect a "tear drop" distortion from the pattern to be expected of radiation from a central focus. The precise nature of the pattern to be expected depends on many variables, but the gene might be expected to occur with a low frequency further to the west than to the east of the area of maximum frequency. The present sketchy data are insufficient for an opinion on this point.

The second approach to the time of origin of the gene depends on detailed anthropological studies of the tribes with the highest frequency of the gene. If they are otherwise essentially no different from the surrounding tribes, this suggests a post-migrational origin. But if they are set apart by characteristics which, for example, link them with older human strata, then one is forced to visualize a migratory current which swept around and by them. Returning to figure 4, we note an approximate correspondence between the area of highest frequency for the hemoglobin C gene and the third of the areas in West Africa in which "paleonegroid" elements are prominent. This latter is the "Niger circle" area, into the western portion of which there seemed to have been compressed a number of relatively primitive and thus far poorly studied tribes.[3] However, although the coincidence is certainly apparent, it should be emphasized that the area is large and that only a few of the tribes involved have been sampled thus far. The present evidence, then, is at least consistent with the view that the origin of hemoglobin C may well be pre-migrational with respect to the sickle cell gene—but sufficiently recent that the gene had not yet had the opportunity to diffuse extensively throughout the "paleonegroid" tribes of West Africa. An alternate possibility, based in part on the evidence that a number of the tribes of West Africa show a complementary relationship between the frequencies of the S and C genes, with the appropriately weighted sum of the two approximating a constant (Allison, '56), would be that the C gene had arisen post-migrationally and is in the process of displacing the S gene. The differentiation between these two possibilities rests in large part on intensive study of the little known primitive tribes of the Niger circle. If, in tribes which by all standards have the smallest infusion of genes from later migrants into West Africa, the sickle cell gene is either absent or present in low frequency while the C gene is relatively frequent, the most logical explanation would seem to invoke an origin of the C gene prior to the introduction of the S gene. This, of course, does not rule out the possibility that where the two genes overlap in their distribution, C will now displace S.

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[3] I am indebted to Dr. Frank Livingstone for a great deal of helpful discusssion concerning the anthropology of West Africa.

One further development should be mentioned. With the conditions under which paper electrophoresis is usually carried out, most of the abnormal hemoglobins migrate more slowly than normal, but American investigators have recently described three new and rapidly migrating hemoglobins, each on the basis of the findings in a single family. These are termed H (Rigas, Koler and Osgood, '55), I (Rucknagel, Page and Jensen, '55) and J (Thorup, Itano, Wheby and Leavell, '56). We have found in Liberian natives two rapidly migrating hemoglobins, one occurring in 7 out of 920 individuals, the other in two out of 920 individuals (Robinson, Zuelzer, Neel, Livingstone and Miller, '56). The more frequent of these corresponds to hemoglobin J, which in thousands of electrophoretic studies on randomly selected American Negroes has thus far been encountered in only one individual. The other hemoglobin appears to be distinct from both H and I, and hence to be a new one. Independently of the American investigators, Cabannes and his collaborators have described two rapidly migrating hemoglobins in blood specimens from Algerian natives (references in Cabannes, Duzer, Portier, Massonnat, Sendra and Buhr, '56). Neither of these corresponds to hemoglobin H, but their relation to I, J, or the new hemoglobin encountered in Liberia is not clear. In a sample of 1,624 hospitalized Algerians studied by Cabannes and his collaborators, there were 4 individuals with one of the fast components and one with the other, while 0.9% had hemoglobin

C either alone or in combination with A or S, and 0.5% had hemoglobin S, alone or in combination with A or C (Cabannes, *et al.*, '56). The finding of S and C in these relative proportions suggests that the Negro admixture in the North African native may, on the basis of the hypothesis discussed above, involve a higher proportion of the paleonegroid stock than of the more recent arrivals. Furthermore, if the fast-moving hemoglobins encountered in North Africa prove to be the same as were found in Liberia, then it may be postulated that these hemoglobins will in time be discovered in some of the tribes of the French Sudan, a finding of importance in understanding the biological relationships of the tribes of this area. Thus, since Cabannes *et al.* encountered fast-moving hemoglobins most frequently in the Kabyle Berbers, we may speculate that these hemoglobins will be found in the Tauregs and related peoples of the French Sudan; if the fast hemoglobins encountered in Liberia prove similar, a very interesting pattern of gene exchange in prehistoric times will emerge. In this connection, it should be noted that in the tribe with the highest proportion of individuals exhibiting hemoglobin C yet studied (26.8% in 71 northern Gold Coast Dagomba; Edington and Lehmann, '56), no examples of a rapidly migrating hemoglobin were encountered.

Finally, on the assumption that the malaria hypothesis supplies the principal explanation of the high frequency of the sickle cell gene, let us consider briefly the consequences of malaria control in Africa. Such control must lead to a decrease in the frequency of the gene, in a perfectly predictable manner worked out by Smith ('54). For the time being, this is probably the clearest example in all human genetics of the effect of an environmental change on the future evolution of man.

The abnormal hemoglobins represent but one of a number of situations where there are marked gene frequency differences between racial groups. The findings regarding the various inherited serotypes at once come to mind. Although at one time there appeared to be a sharp difference in the population genetics of the "unfavorable" hemoglobin genes and the more nearly "neutral" serotype genes, we must now, as I pointed out a few years ago (Neel, '54), recognize a continuity of problem. There are decreasing grounds for regarding the situation typified by the hemoglobin genes as a genetic curiosity, clearly to be distinguished from the more orthodox behavior of the genes producing serological reactions. Rather, in the hemoglobin genes we are confronted with a tactically favorable situation from which may emerge answers of universal applicability. With respect to quite a number of human genes which, because of their frequency, distribution, and clear-cut effects are favorable material for studies on selection, we find ourselves in something of the same position as virologists interested in the large group of adenoviruses. These are sometimes called the " orphan " viruses, since so many of them have not yet been related to specific diseases. So, also, for many of the known human genes we have yet to acquire an appreciation of the range of their effects. If recent studies on the relation between ABO blood groups and duodenal ulcer are correct in suggesting that an important factor may be the blood group of the mother of the affected individual (Clarke, Edwards, Haddock, Howel-Evans, McConnell and Sheppard, '56), this provides an interesting insight into the complexity of some of these selective relationships. Further insight into the undoubted complexity of these problems is provided by the relationship of ABO blood type to the probability of Rh isoimmunization (Levine, '43; Van Loghem and Spaander, '48) and the role of the secretor factor in the development of duodenal ulcer in a blood group O individual (Clarke, *et al.*, '56).

SUMMARY

We have briefly surveyed three areas in which the time seems propitious for work on natural selection in human populations. Other areas will undoubtedly occur to the reader. The study of the means whereby human populations rid themselves of undesirable genes and conserve those advantageous to the species is scarcely underway. Not until we are in command of such information can we truly understand man's past or predict his future in a rapidly changing world.

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SOMATOTYPE AND SEXUAL MATURATION IN BOYS: A METHOD OF DEVELOPMENTAL ANALYSIS

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IN a previous paper, the senior author mentioned the desirability of describing the typical trajectories of physical growth and development of individuals of different body builds (Hunt, '52). In the present study, such trajectories have been calculated from data on a succession of stages of sexual maturation in boys. These findings were obtained during a growth study under the direction of one of us (J. R. G.), which was carried out from 1939 to 1942 on a group of boys attending a boarding school in New England. Information was utilized on their ages, somatotypes, and degrees or stages of sexual maturation. As used here, the data are wholly cross-sectional.

In order to arrive at these developmental trajectories, the senior author devised a chronological method of describing sequences of stages of human maturation. This procedure can be used whenever ratings of somatic development are available for a sample of children of various ages. The technique is based on elementary concepts of statistics and logarithms and has both versatility and generality. One can readily compute the variability of a group of children in attaining the stages of maturation, a developmental age equivalent for each stage, and at any desired age, the incidence of children in the population who are within the different stages.

If stages of development have been assessed for several distinct tissues or organs in a group of children, this method makes it possible to determine the degree of integration or unity of maturation of these structures, even where the sequence of appearance of the stages may vary from one child to another.

Within a total series of children, this procedure also permits the comparison of subgroups with one another. The chronology of the total series is considered to be normative with respect to variability, earliness or lateness of maturation (timing), and the velocity of passing through the stages. By further calculations, these three measurements of each subgroup are compared with those of the total series. Alternatively, these three measures from two separate samples of children can be compared with one another.

Variability, timing and velocity add up to a relatively complete description of a developmental trajectory in a group of children. In the present study of physique and sexual maturation, data of this kind seem to indicate a mechanism at the basis of sexual development which might well be studied experimentally.

The Theory of Normative Developmental Chronologies

For the entire series of boys, the chronology of their sexual maturation was based on what we call *attainment curves*. An attainment curve is a distribution representing the percentages of individuals who have *attained or surpassed* any particular stage of maturation, as plotted against either their chronological or their developmental ages.

Even the simplest graphic attainment curves often yield insights into growth and development. Notable in this respect are studies of the attainment curves of the emergence of teeth in man by Hurme ('48) and in the rhesus monkey by Hurme and van Wagenen ('53, '56). "Emergence" in this case refers to the brief period during the eruption of a tooth when its crown first perforates the gingival mucosa.

In general, such emergence curves are positively skew cumulative frequency (ogive) distributions, as shown in figure 1a. The data for figure 1 are based on the emergence of the deciduous mandibular central incisor in Boston White infant girls, as published by Doering and Allen ('42).

Because of the skewness of these distributions, the median age of emergence of a tooth is somewhat younger than the mean. Furthermore, the age range from the first percentile to the median, for example, is shorter than the interval from the median to the 99th percentile (Hurme, '48). It is therefore desirable to modify the widths of the class intervals of the age axis in order to eliminate this skewness.

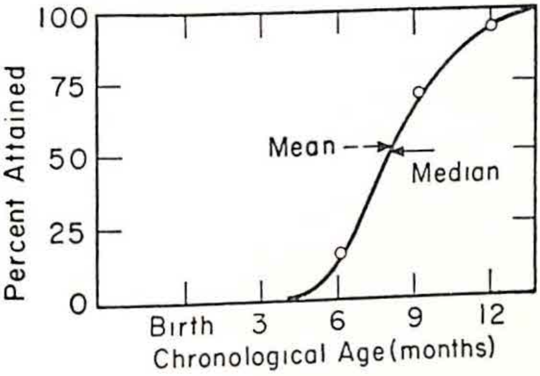

FIG. 1A. ORIGINAL ATTAINMENT CURVE (SKEWED).

Kihlberg and Koski ('54) found that when the age axis is transformed into a logarithmic scale, such skewness disappears because the lower half of an attainment curve is stretched along the age axis, and the upper half is contracted. As shown in figure 1b, a semilogarithmic plot of an attainment distribution produces a symmetrical ogive. As will be seen presently, this new curve is also normal.

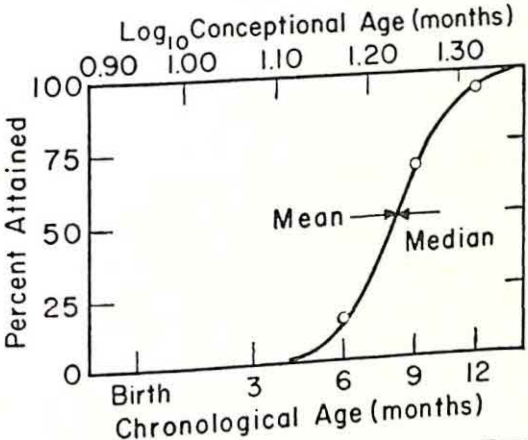

FIG. 1B. AGE AXIS TRANSFORMED AND SKEWNESS ELIMINATED.

For most purposes, the most suitable logarithmic transformation of age scales is the approximate interval of time from conception to the date of observation (Kihlberg and Koski, '54). In man, this so-called "conceptional age" is usually taken as age from birth plus the traditional average human gestation period of 9 months. A slightly more accurate estimate of the mean gestation period would be 8.9 months, or 0.74 year (Hotelling and Hotelling, '32). This value is used in the present study. Where the true date of conception is known, however, the exact conceptional age can be used (Hurme and van Wagenen, '56).

In any logarithmic transformation, the earliest log cycles have a total breadth which approaches infinity. In studies of infants, it is therefore undesirable to consider birth as the origin of coordinates of the age axis and far better to use conception. By this device, the problem of inordinate breadths of the early log cycles occurs only before the blastocyst is implanted in the uterine wall. The scale of log conceptional age makes it easy to study the continuity of fetal and postnatal growth.

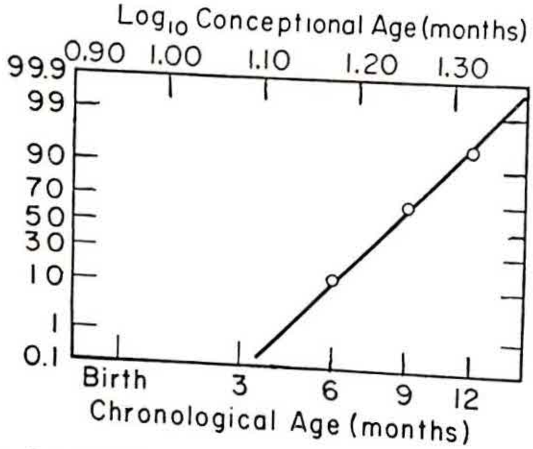

FIG. 1c. PERCENTILE AXIS TRANSFORMED (LOG PROBABILITY GRAPH) TO PRODUCE LINEAR ATTAINMENT CURVE.

For the convenience of the reader, it is often desirable to label the postnatal intervals of this scale by chronological age. Jennings and Pyle ('55) have used such a relabeled scale extending from birth through adolescence in their "Merrill-Palmer logarithmic developmental graph." This graph can be used to relate developmental and chronological ages in individuals or groups of children.

The S-shape of a normal ogive plot of percentiles on log conceptional age can be transformed into a straight line if the upper and lower ranges of percentiles are properly stretched at the extreme values (figure 1c). In precise terms, consider a scale where the 50th percentile equals zero, and which is positive at higher percentiles and negative at lower ones. The percentiles are converted into standard deviation units by means of a table of areas under the normal curve. For example, the standard score + 1.00 is at percentile 84.13, and — 1.00 is at the 15.87th percentile.

The correct spacing of the percentiles and log age units are built into the so-called "log probability" graph paper, which may be purchased from the Codex Book Co., Norwood, Mass. On this paper, the user records the chronological age units at intervals on the log axis for conceptional age. He then plots the percentages of individuals in various age groups who are at or beyond a given stage of development and connects these points with a line, as shown in figure 1c.

A succession of linear attainment curves can readily be drawn on one graph, and the line for the terminal stage characterizes only the *attainment* of this final level of development. If interest centers on the maturation of several distinct organs, such as different classes of teeth, attainment curves from these diverse sources can be plotted on one graph.

If consideration is limited to one developing organ, its family of attainment lines must *necessarily* be parallel if all the stages are clearly separable and irreversible in their sequence. If a line were not parallel with its neighbors in the sequence, it would intersect with them, and at percentiles beyond these intersections, the stages would be erroneously reversed.

At the Forsyth Dental Infirmary for Children, Miss E. A. Fanning has made extensive tests of this expectation of parallelism in radiographic studies of dental development. This statistical phenomenon occurs in the stages of calcification of a tooth, and also in the stages of resorption of roots of deciduous teeth.

An important test of parallelism is whether it occurs in the development of separate tissues or organs. Where it does exist, these structures apparently mature in a systematic or integrated relationship with one another, but not necessarily in an irreversible sequence. The further exploration of such a system would involve correlations between the standard scores of attainment in the development of the different organs, and tables of the incidences of the various alternative sequences of stages in the population.

The occurrence of parallelism in the development of several organs seems to have been first demonstrated by Kihlberg and Koski ('54). These authors found that different classes of teeth in some instances exhibit parallelism in their curves of clinical emergence in both man and the rhesus monkey. We have made plots of the succession of developmental stages of pubic hair and genitalia in boys, as published by

Reynolds and Wines ('51), and found that the attainment curves of these two tissues are parallel. The same is true of the three attainment lines for initial breast development, first pubic hair and menarche in girls, based on data from Reynolds and Wines ('48).

An algebraic treatment of parallel attainment equations reveals some of their very useful statistical properties. Let c represent a positional constant which is distinctive for the equation of each stage in a succession. For all the equations of the sequence, z represents the standard score of the percentile of individuals at or beyond a given stage, t equals conceptional age, and a equals a constant. All the parallel lines will then assume the form:

$$z = a \log t + c.$$

All of the attainment equations will have identical slopes, and this slope is also the standard deviation of each attainment distribution in units of log conceptional age: i. e.,

$$\sigma_{\log t} = \frac{1}{a}.$$

In an irreversible sequence of parallel attainment lines, let us determine what "developmental age equivalents" should be assigned to stages which are transitory or intermediate states of the organism. For any such stage, we define a segment of the log t axis, extending from the mean age of attainment of the stage to the corresponding mean of the next succeeding stage. The midpoint of this segment is the "midpoint age."

Consider a theoretical population, all members of which are at the midpoint age. Half of these individuals will be *in the middle of, or beyond* this developmental stage. Still more important, at this age, the incidence of individuals *at or within* the stage is maximal. Thus, the midpoint age is simultaneously a mean, median and mode, and is therefore a proper developmental age equivalent of the stage.

In publishing growth data, one may need a frequency distribution of individuals who are within a stage. For this purpose, we merely require a bell-shaped normal distribution with a mean at the appropriate midpoint log conceptional age and a logarithmic standard deviation of $1/a$.

The maximal incidence of individuals at the midpoint age is calculated by substituting the midpoint age in units of log t into the equations of the two adjoining attainment lines and solving each equation for z.

These two standard scores are then converted into percentiles, and the percentile for the later stage is subtracted from that of the preceding one.

Calculations of incidences can of course be produced for any desired age. To find the incidence of individuals who have not yet entered the sequence, we subtract the percentile of the least mature stage from 100%. The percentile for the terminal equation gives the incidence at this age who have completed the sequence. The difference between the percentile of attainment of any intermediate stage and the next succeeding percentile represents the incidence within the stage. By such steps, it is easy to prepare a table of incidences for any desired set of ages from a log probability plot.

The Theory of Group Comparisons

In considering the pattern of development in an arbitrary subgroup taken from a total series of individuals, we calculate a family of attainment equations for the subgroup alone. In this case, the standard deviation of ages of attainment ($\sigma_{\log t}$) of the subgroup is compared with the corresponding normative standard deviation for the total series. Such a comparison indicates whether the subgroup is more or less variable than the total series in its maturation.

For the additional comparisons of the subgroup with the total series, we employ the method of allometry. In a series of organisms which vary in size and shape, allometry is the use of equations which portray the relationships between the logarithms of two somatic measurements or biochemical determinations (Smith, '54; Hunt and Giles, '56). In the present study, however, the method involves the comparison of two sets of mean logs of conceptional age of attainment of a sequence of maturational stages. For our allometric equation, y represents the mean conceptional ages of attainment for the total series and x those of the subgroup. Log b is a positional constant and k is the coefficient of relative growth. The equation is in the form:

$$\log y = k \log x + \log b.$$

Since this equation characterizes an allometry of time rather than of metrical data, the constant $\log b$ is not a mere artifact of the units of measurement. Instead, the sign and value of $\log b$ help to show the *timing* of sexual maturation in the subgroup. Where $\log b$ is negative and k is close to unity, the subgroup is consistently late in maturation. Where k is close to unity and $\log b$ is positive, a subgroup is consistently early in maturation. Where k diverges far from unity, however, the interpretation of k and $\log b$ is likely to be more complicated.

The k coefficient measures the *velocity* of maturation typical of the subgroup. Where $k = 1.25$, for example, the subgroup has traversed its developmental sequence 25% faster than the total series. If $k = 0.75$, on the other hand, the development of a subgroup is only 75% as fast as the average for the total sample.

As will be seen later, a satisfactory computational technique for the present data remains for the future, and as used here, the comparisons are admittedly crude. The biometrician will see that confidence limits and tests of significance for the various statistics of the attainment curves would be highly desirable.

The testing of parallelism in an assortment of attainment equations is not unlike the evaluation of parallelism in the slopes of equations of dosage responses in bioassay investigations (Bliss and Calhoun, '54). The closest analogy is the replication of assays of a single unknown biological substance as compared with a standard substance. The simplest fiducial test of parallelism of attainment curves would be based on cross-sectional data such as the present materials on adolescent boys. Adapting the test to semi-longitudinal or fully longitudinal data, however, would probably not be easy. A beginning might be made in such cases along the lines suggested by Tanner ('51) for metrical data.

The present method of comparison need not be limited to a subgroup as related to a total series. An even more important application might be the comparison of two or more distinct groups of individuals—especially when one group served as a reference or normative standard of development. On the basis of the normative sample, the variability, timing and velocity of development of the other groups could be conveniently recorded and interpreted.

The Calculations for the Total Series

Greulich *et al.* ('42) published data on the percentages of boys who were within a series of stages of maturation of the genitalia and body hair in age classes ranging from 12 to 17 years. These findings were based on 476 boys from the same boarding school as the smaller series used here. On log probability paper, we found that the attainment curves for these stages were straight parallel lines. These lines were not fully precise, however, because the midpoints of the age-class intervals were not specified.

The basic stages of maturation are 5, and a boy can be assigned either to a basic stage, or to an intermediate stage such as 1-2, 2-3, 3-4 or 4-5. This elaboration gives a full complement of 9 stages. The 5 basic stages are described in detail by Greulich *et al.* ('42). In brief, they are as follows:

1. External genitalia small as in a young child; body hair unpigmented vellus only.

2. Testes usually enlarged; long, downy, slightly pigmented hair present at base of penis.

3. Penis definitely enlarged; first coarse, fully pigmented pubic hair; often first lightly pigmented facial hair.

4. Further enlargment of penis and testes; pigmented hair not yet present on thighs or lower abdomen; coarse facial hair present, especially on upper lip, in front of ears, and chin. Circumanal and axillary hair usually well developed.

5. Penis and testes at nearly adult size and sometimes disproportionately large. Public hair usually as in the adult in quantity and type. Coarse pigmented hair present in hairy individuals on thighs and linea alba. Variable amounts of coarse terminal hairs on upper lip, chin, and sides of face. Circumanal and axillary hair well developed.

These stages were rated by personal inspection of the individual boy by Greulich.

Of the 476 boys in the entire growth study described by Greulich *et al.* ('42), 226 had not only been assigned to the preceding basic or intermediate stages, but had also been somatotyped by C. W. Dupertuis. The ratings of physique were performed from body build photographs according to the inspectional technique of Sheldon *et al.* ('40).

The present authors utilized this lesser sample in computing a normative set of parallel linear attainment equations. The earliest equation is for boys at or beyond the onset of pubescence (stage 1-2), and the 8 remaining equations apply to the subsequent basic and intermediate stages.

The 226 boys ranged in chronological age from 13.08 to 17.58 years when their maturation was rated. Within two months of this occasion, each boy was photographed for somatotyping.

For computational purposes, the series was separated into younger and older groups at the 15th birthday on the basis of the age at which maturation had been rated. For each group, the percentage of boys at or beyond each stage was found. These percentages were then converted into standard scores from a table of areas under the normal curve.

For the time axis, the age when each boy was given his maturational rating, was converted into the $\log_{10}$ of conceptional age. For the younger and older age groups, the means of the logs of conceptional age were found. The two means are the age-coordinates for the arrays of standard scores of attainment.

In order to find the common slope or standard deviation of the attainment curves, a bivariate distribution was set up of all the standard scores of percentages of attainment within the limits 0 — 100%. The stages within these limits in both the younger and older groups were 3-4, 4, 4-5 and 5. A least-squares regression of z on $\log t$ was computed to arrive at a value of a for these 4 stages. The reciprocal of this value was then the common slope or standard deviation ($\sigma_{\log t}$) for these 4 stages. The remaining step was to calculate c for each separate attainment equation.

In the younger group, percentages of less than 100% were available for stages 1-2, 2, 2-3 and 3. $\log t$ was fixed at the mean age for the younger group, as described earlier, and a was the constant which had been derived in the preceding least-squares computations for the later stages. Substituting the appropriate z score for each early stage into the basic linear equation along with $\log t$ and a, the corresponding value of c was found.

For stages 3-4, 4, 4-5 and 5, the values of c were computed by substituting the constant value of a into the two normal equations which are customarily used for least-squares estimations. Where N represents the number of z scores used (two in all the present cases), the first normal equation was

$$\sum z = Nc + a \sum \log t.$$

This value of c characterizes an attainment equation which crosses the centroid of the two loci of the z scores.

The second estimate of c, from the other normal equation, is the least-squares line of regression of z on $\log t$. This equation is

$$\sum z \log t = c \sum \log t + a \sum (\log t)^2.$$

As shown in table 1, the centroid and least-squares estimates of c agree closely except for stage 4-5. This agreement is indicative of the suitability of the system of parallel lines as a fit for the present data, and the mean of the centroid and least-squares estimates for each equation is used for the later computations in this paper.

Calculations for the Somatotype Subgroups

The 226 boys in the present series were divided into 6 subgroups according to their dominance of one, or balance of two or three, of the somatotype components. For each subgroup, parallel attainment equations were computed as for the total series. One small subgroup (the balanced endo-mesomorph class) had to be omitted from consideration because its younger array was more advanced in maturation than its older one. All that could be done with this omitted subgroup was to find the incidence of stages at the mean log conceptional age of the whole undivided class.

The values of a for the large samples of dominant mesomorphs and dominant ectomorphs were based on stages 3-4, 4, 4-5 and 5. The smaller samples with balanced mesomorphy and ectomorphy, and with equality of all three components, have this coefficient based on stages 4, 4-5 and 5. The smallest usable sample (the endomorphs) has a value of a based on stages 4-5 and 5 only.

As in the total series, the values of c for some of the earlier stages were found from the value of a for the combined later stages, together with the appropriate computations of z and $\log t$ for each younger array of boys. For the separate later stages, the centroid and least-squares values of c were calculated as for the total series.

For each subgroup, the standard deviation ($\sigma_{\log t}$) expresses the degree of individual variation in attainment of the stages. The log t-intercepts of its family of attainment curves provide a set of mean log conceptional ages which can be compared with their equivalents for the entire series by the method of allometry.

Each of the allometric equations was computed by the simplest reduced major axis method of Kermack and Haldane ('50) from the mean conceptional ages of attainment for the total series (y) and the corresponding means of each somatotype subgroup (x). Letting $n =$ the number of pairs of means utilized:

$$k = \frac{\sigma_{\log y}}{\sigma_{\log x}} \text{ and } b = \frac{\sum \log y - k \sum \log x}{n}.$$

FINDINGS ON THE TOTAL SERIES

The chronology of attainment of the maturational stages of the total series is shown in table 1, and graphed in figure 2.

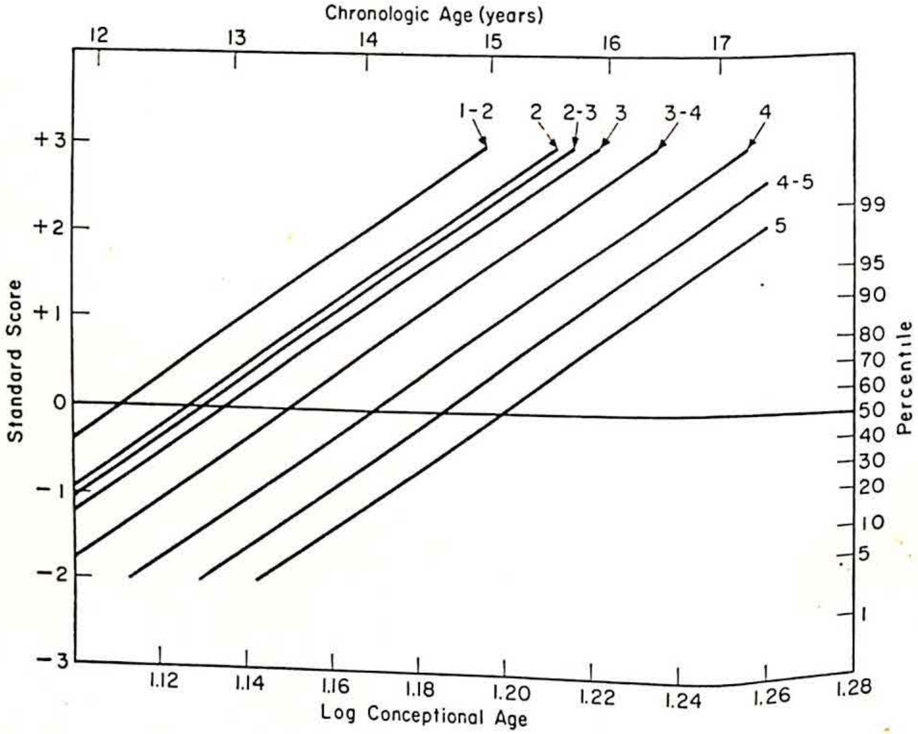

FIG. 2. ATTAINMENT CURVES FOR SEXUAL MATURATION OF 226 PREPARATORY SCHOOL BOYS.

In these boys, the variability of attainment of the stages ($\sigma_{\log t}$) is 0.029 log units. We have analyzed attainment curves from longitudinal data published by Reynolds and Wines ('51) on 4 stages of pubic hair and 4 of the genitalia in boys from the Fels Institute growth study at Yellow Springs, Ohio. All 8 of these equations, too, are parallel and have a variability of attainment of 0.029 log units. This agreement indicates the soundness of the present computational method, even though it is based on cross-sectional data on a restricted portion of the age range of normal adolescent development.

The mean of the first attainment curve in our sequence (stage 1-2) or the onset of pubescence, is at 12.20 years. This estimate is slightly later

TABLE 1

Attainment equations for stages of sexual maturation in 226 preparatory school boys

STAGE	c			LOG t-INTERCEPT (50TH PERCENTILE)			MEAN CHRONOLOGICAL AGE IN YEARS		
	One locus	Centroid	Cross-products	One locus	Centroid	Cross-products	One locus	Centroid	Cross-products
1 — 2	— 38.6786			1.1115			12.20		
2	— 39.2486			1.1279			12.69		
2 — 3	— 39.3536			1.1309			12.78		
3	— 39.5286			1.1360			12.94		
3 — 4		— 40.0174	— 40.0153		1.1500	1.1500		13.39	13.39
4		— 40.6974	— 40.6989		1.1700	1.1700		14.05	14.05
4 — 5		— 41.2424	— 41.2011		1.1852	1.1840		14.58	14.54
5		— 41.7374	— 41.7375		1.1994	1.1994		15.09	15.09

z = standard score of % boys at or beyond stage.

t = conceptional age in years.

Basic equation: $z = 34.7973 \log t + c$.

than the onset of genital maturation in Tanner ('55), but close to the onset of pubic hair development. This finding suggests that on a cross-sectional basis, an observer can judge the initiation of pubic hair development more accurately than the first enlargement of the testes. Pubic hair is either visible or absent in a boy: whereas an observer may be uncertain whether the testes have begun to enlarge unless he has a recent prior body build photograph of the same boy as a standard of reference.

Greulich's stage 3, which includes the first appearance of coarse and fully pigmented pubic hair, is attained at a mean age of 12.94 years in the present series and at just over 13 years in the compilation of Tanner ('55). This agreement is again quite satisfactory.

The final stage in Greulich's chronology, stage 5, is not the true end point of sexual maturation. It is "open-ended," and the mean age of attainment is 15.09 years. This age is much younger than the terminal stages of sexual development as found from serial photographs and personal inspections of boys, especially those of Stolz and Stolz ('51) and Reynolds and Wines ('51). We see no reason, however, to consider this divergence as anything but an artifact in the definition of stage 5. Phenomena such as the shrinkage of the penis in the late teens, as documented in many boys by Reynolds and Wines ('51), would be very difficult to rate on a cross-sectional basis.

FINDINGS ON PHYSIQUE AND MATURATION

The statistical data relating somatotype and sexual maturation in the present study are presented in table 2, and in graphic form in figure 3.

The *timing* of maturation is consistently early in dominant mesomorphs, as noted by Tanner ('55) and others. This finding is nearly as true of the balanced mesomorphic-ectomorphic subgroup as of the heavier muscular individuals. As mentioned earlier, the balanced endomorphic-mesomorphic subgroup was unsuitable for a full mathematical treatment, but of the 15 boys in this small sample, 67% were at stage 5, while the predicted incidence at the equivalent mean log conceptional age in the total series of 226 boys would be 46%.

The *variability* of maturation in the somatotype groups shows a fairly consistent association with the percentage of fat in the body. The leanest group (ectomorphs) have a low variability. The groups with midrange fat content are in the midrange of variability, and the most adipose boys

TABLE 2

Variability and allometric constants of sexual maturation in somatotype subgroups

GROUP	N	VARIABILITY OF ATTAINMENT OF STAGES $\sigma_{\log t}$	ALLOMETRIC CONSTANTS COMPARING SOMATOTYPE SUBGROUP WITH TOTAL SERIES k	b
Total series	226	0.029	——	——
Dominant endomorphs	16	0.033	0.822	+ 0.207
Dominant mesomorphs	91	0.032	0.932	+ 0.085
Dominant ectomorphs	59	0.021	1.442	— 0.082
Balanced, endo- and mesomorphy	15	——	——	——
Balanced, meso- and ectomorphy	19	0.028	0.872	+ 0.155
Balanced, all 3 components	26	0.026	0.968	— 0.033

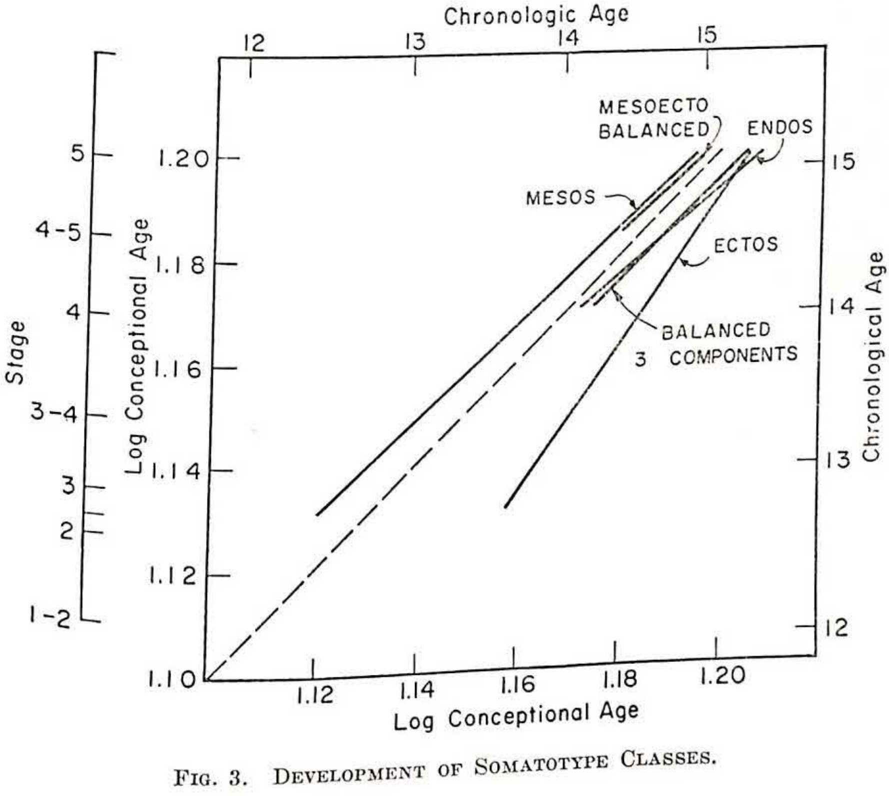

FIG. 3. DEVELOPMENT OF SOMATOTYPE CLASSES.

(endomorphs) are the most variable. This finding agrees with the clinical observation that deviant chronologies of sexual maturation are most often found in obese adolescents of both sexes.

Like variability, the *velocity* of sexual maturation seems to have a relationship to the fat content of the body, but in an unexpected direction. Although the endomorphic sample is small enough to be somewhat suspect, it appears that the slowest maturation occurs in the fattest boys. The velocity is definitely fastest in the leanest sample. The other groups do not deviate much from the average velocity.

Because of his slowness of development, the average endomorph begins sexual maturation early, but completes the sequence of stages late. Because of his very rapid development, on the other hand, the typical ectomorph is more retarded at pubescence than later on.

DISCUSSION

Mesomorphy is easy to see in the external form of the body—especially in males—as the outlines of muscles and usually a relatively masculine appearance. Boys with considerable mesomorphy appear to have an early but rather variable onset of pubescence, and to proceed at about the average velocity through their later stages of sexual development. One can speculate as to whether the tissues of the mesomorphic body respond early and fully, but not rapidly, to androgens, since many of its features appear to exaggerate the ordinary physiologic response to these hormones —particularly the coarse skin and prominent muscles.

Many observers have noted that caloric nutriture may hasten or retard the maturation of the mammalian body (Tanner, '55). In our opinion, caloric balance is important in influencing the time of *onset* of sexual maturation. If a boy consumes more calories than he needs, he will grow fatter and in so doing may reach pubescence sooner.

Caloric nutriture, as reflected in total body fat, also seems to influence the *velocity* of sexual maturation, but in a manner different from its effects on the beginning of pubescence. The progress of the maturational stages is at least partially controlled by androgens and other hormones. Once the transformation has started, it appears that the rising titers of these hormones produce an intense response, or rapid maturation, in the lean individual. This reaction is rather reminiscent of the physiological response to stimulant drugs in ectomorphs.

Since the steroid hormones are fat-soluble, one wonders whether they are stored or metabolized in the excess adipose tissue of fat boys, and are thereby prevented from exerting their full effects on the maturing genitalia and sexual hair.

These conjectures emphasize the desirability of further research—especially on a longitudinal basis—on body composition, the titers of endocrine secretions in body fluids, and rates of maturation in adolescents of both sexes. Another desirable project would be to learn how fat animals differ from lean ones in their sensitivity to steroid hormones, as well as in their transport, storage and metabolism of these substances.

SUMMARY

Where stages of maturation have been defined and applied to a sample of children, it is convenient to record the percentages of individuals who are at or beyond a stage as related to their ages. Such distributions may be transformed into a so-called "attainment curve." If grouped data are available, a convenient method is to plot the percentages on log probability paper. In this case, age is transformed into the logarithm of conceptional age, and the percentages of individuals into standard scores around a zero point at which 50% of the population are at or beyond the given stage.

In studies of both dental development and sexual maturation, attainment curves on log probability paper form straight parallel lines. For stringent theoretical reasons, a succession of attainment curves for stages of development within one organ tend to be parallel. Tests of parallelism between different organs are valuable indications of their systematic or integrated relationships with one another.

This method of recording the chronology of developmental stages has versatility and generality. It can conveniently be attempted whenever data are available on children of various ages and degrees of maturation. With little computational effort, the method can provide an abundance of information such as the variability of the group in attaining its stages of development, a developmental age equivalent for each stage, and at any desired age, the incidence of children in the population who are within each stage.

A crude method was devised of computing the equations of parallel attainment curves. This procedure was applied to the stages of sexual maturation which had been recorded in 226 boys at a New England boarding school. These students had also been somatotyped. A study was therefore carried out which related body build and the trajectories of sexual maturation in these boys. For each of 5 somatotype subgroups,

we calculated the variability of attaining the stages. Allometric equations relating most of the subgroups to the total series revealed the timing and velocity of maturation in boys of different physiques.

Our data agreed with the findings of many previous authors that mesomorphy is associated with early maturation. In predominantly mesomorphic boys, the attainment of the stages is slightly more variable that the average, and the velocity of maturation is about average.

The association of ectomorphy with late maturation has long been known. In our group, ectomorphs show a small variability in the ages of attainment of the stages, but their velocity is 44% faster than the average. This rapidity of development is suggestive of a physiologic over-response to the increasing production of androgens and other trophic hormones which occurs in adolescence.

Although the endomorphic sample is small, it shows high variability in the ages of attainment of the stages, an early onset and late ending of sexual maturation. The velocity is therefore low.

Possible relationships between these findings and nutriture and body composition are discussed.

ACKNOWLEDGMENTS

The original study of the school population was supported financially by the Carnegie Corporation, and the present analyses were carried out in part with the support of The Grant Foundation, Inc. The methodology and machine computations were developed during the senior author's tenure of a Fulbright Visiting Lecturership at the University of Melbourne, Australia.

The authors especially wish to acknowledge the help of C. W. Dupertuis and W. W. Greulich, whose ratings of physique and maturation are the basis of the entire investigation. Others who have contributed much to this project have been A. F. Roche of the Department of Anatomy, Melbourne; R. C. Reed and J. Mayer of the Harvard School of Public Health; and W. W. Howells of the Harvard Department of Anthropology.

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BOOK REVIEWS

Modern Ideas on Spontaneous Generation. Edited by R. F. NIGRELLI. 121 pp. Ann. N. Y. Acad. Sci. *69*, Art. 2. N. Y., 1957. $2.50.

If the title of this symposium seems startling to the reader, let us admit that it also seems so to the reviewer and evidently even to some of the contributors. Blum, in his introductory remarks, mentions that the term " spontaneous generation " was taboo among biologists only a few years ago. Of course the modern use of the term has little in common with the ancient pictures of flies arising spontaneously from dead animals and frogs from mud. This is not happening now, and never did. It may be that even the simplest living molecules cannot arise now from non-living material. But H. F. Blum points out that our concepts of evolution and the biochemical evidence we now have seem to require us to believe that, at some time in the past, life did emerge from a non-living milieu.

One of the conclusions forced on the reader is that earlier speculations on the origin of life and the nature of the earliest living molecules are now being taken more seriously than when first proposed. This reviewer bought one of the first copies of the English translation of Oparin's pioneer work. He cannot remember ever discussing it with a scientist who took it seriously. Oparin's ideas seem to be the basis for modern thinking on the subject, and S. L. Miller gives in his chapter of the Symposium an account of his now classic experiments in which he successfully tested experimentally the ideas of Urey and Oparin that organic molecules such as amino acids might originate spontaneously in a reducing atmosphere such as that of the earth in its early stages.

From amino acids to proteins is in theory but a step; the actual sequence of events is still unknown. S. Granick suggests that the first organization of preprotoplasm would be a primitive energy-conversion unit, originating from some common minerals containing metal ions serving as coordinating templates and catalysts, which could perform the elementary process of photosynthesis and respiration. Granick presents interesting suggestions as to the origin of enzymes from inorganic ions by successive complexing with organic compounds.

G. Wald contributes a fascinating discussion of the origin of optical activity, including an apparently original suggestion that from the unit molecules provided by geochemical processes in racemic mixtures, single optical isomers were selected in the process of composing structures of higher order: polypeptides and proteins from amino acids, nucleic acids from nucleotides, etc.

This symposium does not reflect particularly the growing conviction of many investigators that nucleic acid is the primary stuff of heredity and therefore of life, and C. C. Lindegren in his chapter points out the inadequacy of destruction of transforming principles by DNAase to prove that the gene is composed exclusively of DNA.

Space does not permit a summary of the other contributions: S. H. Hutner and others write on "Electrolyte requirements of protists and archeometabolism," A. Gulick on "Phosphorus and the origin of life," R. C. Warner on "The interaction of synthetic polynucleotides," D. Harker on "The structure of crystalline proteins," S. W. Fox and others on "Spontaneous generation of anabolic pathways, protein, and nucleic acid," and N. W. Pirie on "Some assumptions underlying discussions on the origins of life." P. H. Abelson contributes a chapter on "Some aspects of paleobiochemistry" which was of particular interest to the reviewer.

This timely monograph will be of value to all interested in the matter, although as is to be expected from the present state of our knowledge, it is thought-provoking and suggestive rather than definitive or comprehensive.

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Chemical Anthropology: a New Approach to Growth in Children. By ICIE G. MACY and H. J. KELLY. xi + 149 pp. University of Chicago Press, 1957. $3.75.

From 1942 to 1951, Drs. Macy and Kelly published three admirable volumes entitled *Nutrition and Chemical Growth in Childhood.* The present work, *Chemical Anthropology,* is a brief epitome of these earlier studies, with the added aim of calculating age trends in the body composition of children. These trends will be the major topic discussed here.

All four of these books are based on a semi-longitudinal nutritional balance study of healty preadolescent children who resided at the Methodist Children's Village, Detroit. It is still a model of excellence in its field, even though the data were gathered some twenty years ago. Because of its date of inception, hardly any of the modern techniques in the measurement of body composition were used. Instead, Drs. Macy and Kelly have had to use, and sometimes to devise, equations for predicting body constituents from anthropometric data. Some of the trends so obtained have already been published in Human Biol., *28* (*3*) : 289-308. Other constituents which could not be calculated from these anthropometric equations were arrived at by subtraction.

Underlying much of this work is the assumption that the mature percentages of chemical constituents of the human fat-free body are arrived at early in childhood. For a century, it has been known that the fetal organism contains a higher concentration of water than that of the adult. In 1923, Moulton estimated that mature hydration, which is the minimal adult water content of the human fat-free body, is attained in the interval from 500 to 1000 days post conception (J. Biol. Chem., 57: 79-97). Macy and Kelly accept this formulation on the grounds that from the postnatal ages of 4 to 9 years, dimensional growth is relatively orderly and predictable in most children, and the increments of growth per unit of time are also fairly constant. To Macy and Kelly, this linear growth rate is an indication of a stable and even mature chemical composition of the fat-free body.

Logically enough, this interval of linear growth is the straightest portion of a curve of average measurements of a child population as plotted against chronological age. Since this linear interval occurs in many organisms besides man, we may learn from lower forms when this interval takes place relative to the attainment of chemical maturity. The data of Spray and Widdowson (Brit. J. Nutrition, *4*: 332-353) enable us to draw such conclusions from growth curves of body weight and carcass analyses of the rat, rabbit, cat and pig. In all of these species, mature hydration—and chemical maturity in general—are reached *at or after* the end of the interval of linear growth in weight.

Two lines of evidence suggest a similar situation in man. In the laboratory of the late Professor T. Wingate Todd, Venar measured the fresh wet weight and dry weight of children's skeletons. He found that the mature or minimal hydration of the skeleton was not reached until 12 years of age, but felt uncertain about his findings in view of Moulton's much earlier estimates of the age of mature hydration of the fat-free body (p. 92, Part II, White House Conference on Child Health and Protection).

This reviewer and E. Giles calculated the allometry of water and fat-free solids in human fetal and neonatal carcasses, and projected these trends into childhood (Human Biol., *28* (*2*): 253-273). Our estimates of the minimal ages of mature hydration were about 10½ years in girls, and 14 years in boys. These estimates agree quite well with Venar's.

In *Chemical Anthropology*, prediction equations have been taken from the work of Friis-Hansen and his associates (Pediatrics, 7: 321-327) to arrive at total body water from body weight and from surface area. Drs. Macy and Kelly have computed their own equations for thiocyanate space from stature, weight and surface area, based on data from Morse *et al.* (Am. J. Physiol., *151*: 438-447). The values for total body water are of course close to those based on the equation of Hunt and Giles, which also comes from the Friis-Hansen study.

Accepting Moulton's early age of mature hydration, Macy and Kelly utilize a constant water-solids ratio. Since total body water falls from 57% of body weight at 4-6 years to 53% at 10-12, fat content must increase from 22% to 28% in the Detroit boys. If on the other hand the fat-free body continued to "solidify" during this span of growth, fat would be the constant constituent during the linear interval of growth.

It is difficult to reconcile an increasing fat content in preadolescent boys with the decreasing adiposity of the brachium (Baker, Hunt and Sen, Am. J. Phys. Anthrop., in press). Furthermore, studies of body density, total body water, and radiographs and skinfolds of adolescent boys agree that adolescent boys after the age of 12 years are far less adipose than the 28% which Macy and Kelly assign to the 10-12-year-old Detroit group. (Heald, Hunt *et al.*, in preparation).

Aside from these unresolved problems in the growth of body composition in children, *Chemical Anthropology* presents a wealth of material on the nutrition of the young which is of great value.

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Discussions on Child Development. Edited by J. M. TANNER and BÄRBEL INHELDER. Vol. I and II. The Proceedings of the first and second meetings of the World Health Organization Study Group on the psychological development of the child, Geneva, 1953. Tavistock Publications, London, 1956. 240 and 271 pp. Set of 2 vols. $10.00.

Edited verbatim reports of discussions between experts are becoming increasingly common. Eminent examples are the Macy Foundation Symposia and the Ross Pediatric Research Conferences. The organizers of such conferences do an *invaluable* job in bringing together the experts (usually from far and wide) and the idea to bring the reader into the discussion chamber is appealing. After a two-page stimulating presentation by an expert I find myself getting really interested and then one of two things happen: I either say (I whisper to an imaginary colleague), "I wonder what X will say to *that*?" and then X doesn't say it, and I am frustrated. Or when I hope that the speaker will go on and develop his ideas further, someone interrupts and says, "Do you mean, Doctor Y, that you are not in agreement with so and so?" Of course, this is just what did happen, and does happen in informal symposia; and nothing could be more admirable for a whole new train of discussion ensues. But, to read it in print is confusing and I am wondering whether such transactions merit formal publication.

The present two volumes are the result of the World Health Organization's bringing together twelve experts to discuss the biological, psychological, and cultural factors acting on childhood development and formation of the adult personality. In Volume I, Tanner contributes a foundation on somatic growth; Inhelder leads a discussion on the principles of Piaget (present) and Wallon regarding criteria of mental development; Zazzo and Bowlby present discussions on stages of psychological development and psychoanalytic instinct theory. Two papers (sparsely interrupted) by Grey-Walter on cerebral electro-genesis, and Lorenz on comparative behaviorology, with Mead's constant contributions on the application of anthropological techniques to the study of development, make this volume outstanding and important.

Volume II is devoted mainly to discussions on the mechanisms of learning in the human and animals, and to the social and moral aspects of behavior. The first presentation by Grey-Walter is devoted to the fascinating and important (and to me, rather frightening) electronic tortoise, while Bindra, Liddell and Whiting present other headings for discussion. Discussions on films shown by Bowlby and Lorenz also are recorded.

This account does not do justice to the wealth of material found running through these volumes which represent about one-third of the verbatim record. Condensation of such materials is difficult but I cannot imagine how the editors' job could possibly be improved upon.

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The Human Brain. From Primitive to Modern. By A. M. LASSEK. vii + 242 pp. C. C. Thomas, Springfield, Ill. $4.75.

Arthur Lassek, Professor of Anatomy at Boston University School of Medicine and a principal authority on the structure of the pyramidal system, appears to have a dual purpose in *The Human Brain.* Much of the material is a popular account of neural structure. This is accurate and factual, affording captious critics only a few openings, such as the table on page 31 which gives the maximum weight of the male brain as 2000 and the minimum weight of the female as 800 gms.; figures uncorrected for age and body size are misleading. Lassek's purpose is not, however, mere description but "to portray the significance and impact of the long, past environment upon . . . the humain brain, and what it may mean to us. . . ." For such a purpose a much wider field of knowledge than is encompassed by anatomy must be examined, especially as Lassek desires to "make man more understandable and easier to deal with."

Periodically Lassek reminds the reader that "The outstanding fact in the evolution of man is the development of his mind"—the physical substrate of which is implied to be the brain—but as one progresses one finds the same old difficulties turning up. These are trouble in (1) defining exactly what the structural evolutionary advances are, (2) whether the process of structural evolution (as distinct from mere variation) is still continuing or whether it stopped 500,000 years B. C. (*i. e.* with the commencement of cultural evolution) and (3), if it is going on, whether there is any real correlation between it and those inspiring aspects of intellectual development which capture our imagination.

Lassek plays upon the familiar theme of structural difference between *Homo sapiens* and subhuman primates but quietly slips past the troublesome lack of information on the Pleistocene anthropoids. Everyone admits that the nervous system of man and monkey differ and that among these differences is hidden an interesting story. Still, the differences which exist have not been correlated with any specific function not only for the reason that present day average man rides the crest of a wave of culture, which buoys him far beyond his individual capacity of attainment, but also because we have only the most general idea of what the nervous system looked like in the Pleistocene anthropoids or how it functioned.

Once Lassek has assured us that the brain of *Homo sapiens* is in fact different from that of infrahuman primates he catalogues these differences and uniformly comes to the decision that whatever has happened has led to progress or has been immaterial. A greater development of the parietal lobe leads to manual activity and toolmaking we are told but the obvious degradation in the acuity of audition has, according to Lassek, led to the development of cerebral speech. From a consideration of structure Lassek moves directly into an exposition of the psychological aspects of human progress in the historical period. The implication is, of course, that the discussed differences between man and monkey are responsible for that progress. That these two sets of data are separated both in place and time is not clearly revealed and no material is brought forward to lead me to consider the necessity of changing the statement I made in print last year (Mettler, Fred A. 1956 *Culture and the Structural Evolution of the Neural System.* N. Y. The American Museum of Natural History), notably, that " there is no good evidence to support the assumption that man's neural system has undergone any progressive alteration in the direction of greater size or complexity since the middle of the Pleistocene."

Over half of the book is concerned with the psychological aspects of human progress noted above or, as Lassek calls it, the " Evolution of Mind." The " presavage mind," according to the author, was structurally subhuman and functionally acultural. The " savage mind " is identified with a period in which man " relied solely on hunting and fishing." " It can," Lassek says, " be taken for granted that there was a considerable increase in the magnitude of the human brain between the presavage (animal-man) and savage stages." Initial recognition of the ego he places in the savage period. He appears to regard this as a rather abrupt event, likening it to the stroke of a gong. "With this realization, the first mental gong had struck in man's evolutionary time-clock and he was one step away from the mere reflex and animal stage."

The comparative psychologist, uncommitted to the proposition that the ego must, by definition, be a human characteristic and aware of the strong possessive behavior of animal forms, may find it difficult to accept the implication that the development of ego awareness is not a gradual and variable affair. The circumstances and events Lassek has envisioned do not appear to be entirely in accordance with available data or at least lack universal applicability. In his handling of the literature outside his own area of specialization there is some evidence that the author has fallen into the familiar error of giving uncritical acceptance to material of unequal merit and credence to outmoded and questionable sources.

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Behavior Theory and Conditioning. By KENNETH W. SPENCE. vii + 262 pp. Yale University Press, 1956. $4.50.

The non-specialist who happens onto this volume of the Silliman Lectures may well wonder what subject matter they represent. The title suggests something to do with physiology or psychology. Of these, the former is quickly disposed of. The term "conditioning" betrays a link with Pavlov. But whereas for Pavlov the theory of conditioning was nothing but physiology, for Spence it is anything but physiology. On the other hand, there is no mention of ink blots, IQ tests, or even tranquilizers. The talk is all of habit strength and reinforcement; of eyelid reflexes and rats in mazes; of exponential curves and probability integrals. The non-specialist must be assured that all this is indeed psychology, remote though it may seem from the everyday problems of teaching Johnny to read, improving Johnny's personality, or selling Johnny's parents new automobiles.

To understand Spence's book at all, one must realize that it has nothing immediately to do with man's practical problems. Spence makes it clear in his introductory lecture that he and his students have spent years recording eye blinks and observing maze runs with the sole purpose of finding out how various measurements that can be made upon eye blinks and maze runs are influenced by factors in the person's or animal's environment. If there were only a few possible measurements and only a few determining conditions, the outcome of the researches could be summarized in a table and the job would be done. However, there turn out to be many measures, each depending upon many conditions. Consequently, if one shares with Spence a desire to make detailed predictions about how simple responses are learned, to give exact quantitative answers to questions about how the speed or magnitude or probability of a response will be affected by a change in this or that environmental condition, then one must have a theory.

The theory presented by Spence falls squarely in the main line of descent from the doctrines of associationism and conditioned reflexology that have been largely identified with learning theory in this country for several generations. Luckily for the reader, Spence has achieved theoretical progress by simplifying, rather than extending, the elaborate system within which Clark Hull attempted to comprehend the laws of learning. But the tactical streamlining of Hull's program is accompanied by little change in strategy. Order and economy are introduced into the proliferation of experimental laws by two main devices. Firstly, the tendency for any given stimulus to evoke a particular response is represented by a theoretical variable (" intervening variable ") termed reaction potential. Given the current value of the reaction potential, the axioms of the theory should generate predictions concerning the probability, latency, and other more or less directly measurable attributes of the response. Secondly, the determining conditions are grouped into three main classes: those having to do with frequency of past stimulus-response occurrences, with incentive conditions, and with motivating conditions. The basic laws of the theory relate changes in reaction potential to each of these classes of determiners. The bulk of Spence's book is devoted to summarizing previously existing evidence for the particular laws that he has chosen as axioms and to describing many new experiments which serve both to test the laws and to extend our predictive command over simple conditioning situations.

The Silliman Lectures at Yale University have traditionally represented attempts to acquaint a wide scientific audience with technical developments in particular disciplines. Like its predecessors, this volume will require some work on the part of the non-specialist reader but will probably more than repay the effort. And, although less directly, readers of systematic biology, psychiatry, cybernetics, sociology, and perhaps even educational psychology for years to come will owe Spence a heavy debt if his lectures can somehow be forced to the attention of the many writers in those fields who still regard the doctrines of Pavlov and Thorndike as the last word in psychological theory.

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BOOK NOTICES

PHILOSOPHY OF SCIENCE. By Gustav Bergman. xiii + 181 pp. The University of Wisconsin Press, Madison, 1957. $4.75. A chapter, grown into a book, that was initially conceived as introduction to a volume on the logical analysis of psychology. The book retains its orientation toward the behavioral sciences.

AN INTRODUCTION TO THE STUDY OF EXPERIMENTAL MEDICINE. By Claude Bernard. xix + 226 pp. Dover, New York, 1957. $1.50. A new edition of the classical treatise. The work is worth re-reading and re-thinking in 1958, 93 years after its publication. An informative foreword was provided by Professor I. Bernard Cohen.

TEXTBOOK OF HUMAN ANATOMY. By J. D. Boyd, W. E. Le Gros Clark, W. J. Hamilton, J. M. Yoffey, S. Zuckerman and A. B. Appleton. xii + 1022 pp. Macmillan, New York, 1957. $16.50. A new, collaborative textbook motivated by the need 1) to present only the more relevant topographical details, 2) to emphasize the correlation between structure and function, and 3) not to neglect the fact that anatomy is an independent branch of biological science with problems and achievements of its own.

THE IMPORTANCE OF OVERWEIGHT. By Hilde Bruch. x + 438 pp. W. W. Norton, New York, 1957. $5.95. Eating more and exercising less inexorably leads to increase of weight through fat accumulation, but campaigns against obesity "are apt to overlook individual differences and may create more tension for those people who need to overeat as a means of combating anxiety, for self-assertion, or as compensation for unfulfilled ambitions" (p. 58).

REPORT ON THE WORLD SOCIAL SITUATION. By the Bureau of Social Affairs, United Nations Secretariat. vii + 198 pp. Columbia University Press, New York, 1957. $1.75. The *Report*, prepared in cooperation with several agencies of the United Nations (ILO, UNESCO, FAO, WHO), surveys the world population trends, health conditions, food and nutrition, education and conditions of work and employment. The second part deals with problems of urbanization.

AGEING IN INDUSTRY. By F. Le Gros Clark and Agnes C. Dunne. vi + 146 pp. The Philosophical Library, New York, 1956. $7.50. An inquiry, made in Great Britain and based on figures derived from census reports (1921, 1931, 1951), into the employment of older (65 +) male workers in modern industry.

PSYCHOLOGICAL TESTS AND PERSONNEL DECISIONS. By Lee J. Cronbach and Goldine C. Gleser. 165 pp. University of Illinois Press, Urbana, 1957. $3.50. A stimulating, original, technical contribution to the test theory, evolved from a systematic examination of the decision problems for which tests are used, with implications for biological study of individual differences.

PRINCIPLES OF IMMUNOLOGY. By J. E. Cushing and D. H. Campbell. ix + 344 pp. McGraw Hill, New York, 1957. $6.50. Written for graduate students and college seniors majoring in biology. As pointed out by E. A. Kabat in the Foreword, "immunology and immunochemistry, with powerful techniques for recognizing differences among proteins and polysaccharides, provides a tool of enormous versatility for the study of living systems."

THE VISUAL PIGMENTS. By H. J. A. Dartnall. vii + 216 pp. John Wiley, New York, 1957. $6.00. Describes in detail the physical and chemical properties of the photosensitive substances, extractable from the rod-cone layer of retinae, and the methods used to determine these properties.

NONPARAMETRIC METHODS IN STATISTICS. By D. A. S. Fraser. x + 299 pp. John Wiley, New York, 1957. $8.50. Nonparametric methods, many of them of recent origin, are concerned with standard problems, replacing the familiar assumption of normality by general assumptions concerning the distributions. The treatise is intended as a textbook for advanced courses in mathematical statistics.

THE EVOLUTION OF HUMAN NATURE. By C. Judson Herrick. x + 506 pp. University of Texas Press, Austin, 1956. $7.50. This impressive treatise on "psychobiology" grew out of 60 years of intensive and productive study of comparative anatomy of the nervous system, supplemented by wide reading on human behavior and its biological origins.

HYPOTHALAMUS UND THALAMUS. By W. R. Hess. 70 pp. Georg Thieme, Stuttgart, 1956. DM 36. The Atlas, with German and English legends, constitutes the third volume of the author's outstanding contributions to the *Physiology of the Brain Stem.*

CALCIUM METABOLISM. By J. T. Irving. xi + 177 pp. John Wiley, New York, 1957. $2.75. The monograph considers different facets of the problem (ingestion, absorption, utilization, retention, and excretion), including the calcium content of the human body.

NEBRASKA SYMPOSIUM ON MOTIVATION. Edited by Marshall R. Jones. xii + 430 pp. University of Nebraska Press, Lincoln, 1957. The fifth volume in the important series *Current Theory and Research in Motivation,* with topics ranging from physiological mechanisms of motivation to motivational dynamics of language behavior.

AN INTRODUCTION TO GENETIC STATISTICS. By Oscar Kempthorne. xvii + 545 pp. John Wiley, New York, 1957. $12.75. A successful "attempt to present the basic statistical concepts and tools which the genetic research worker needs, with examples from genetics." The detailed presentation of statistical techniques for the study of quantitative inheritance is an outstanding feature.

HUMAN BLOOD GROUPS AND INHERITANCE. By Sylvia D. Lawler and L. J. Lawler. viii + 103 pp. Harvard University Press, Cambridge, 1957. $1.50. A concise presentation, intelligible to the biology student and interested lay reader, of the subject. A chapter is devoted to a historical survey and to the significance of blood groups for human biology.

PREHISTORIC MAN. By André Leroi-Gourham. ix + 119 pp. Philosophical Library, New York, 1957. $4.75. A popular, readable account.

FEAR: CONTAGION AND CONQUEST. By James Clark Moloney. xii + 140 pp. Philosophical Library, New York, 1957. $3.75. Psychoanalyst's cross-cultural approach to the social and medical significance of fear and rage, with considerations of "experimental anthropology," focussed on contrasting the natives of Okinawa (motherly mothering) and Hawaiian Okinawans (transmission of tensions).

BIOGRAPHICAL MEMOIRS, Vol. XXX. By National Academy of Sciences. 409 pp. Columbia University Press, New York, 1957. $5.00. The volume contains, among others, the biographies of John Dewey (elected to the N. A. S. as a psychologist in 1910), biologist F. R. Lillie, and the psychologist L. L. Thurstone (*Multiple Factor Analysis*, 1947).

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A SHORT HISTORY OF ANATOMY & PHYSIOLOGY FROM THE GREEKS TO HARVEY. By Charles Singer. xii + 209 pp. Dover, New York, 1957. $1.75. A reprint, with minor adjustments, of a valuable work originally (1925) published under the title *The Evolution of Anatomy*. Richly illustrated.

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York, 1956. $3.50. A philosopher's "attempt to introduce the reader to those teachings of Ivan P. Pavlov which are pertinent to the fields of psychology and psychiatry."

DEVELOPMENT OF VERTEBRATES. By Emil Witschi. xvi + 588 pp. W. B. Saunders, Philadelphia, 1956. $8.50. The text, designed for premedical students and zoology majors, presents the development of amphibians, fishes, birds, and mammals, including man (pp. 457-551). Profusely illustrated.

SOCIOLOGY IN THE UNITED STATES OF AMERICA. Edited by Hans L. Zetterberg. 156 pp. United Nations Educational, Scientific and Cultural Organization, Paris, 1956. $2.50. This collection of authoritative "trend reports" deals with methods and systematic theory, social groups and institutions (including sociology of knowledge and science), and some social problems (criminology, ethnic relations, mental health).

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GROWTH IN HEIGHT OF TEN SIBLINGS

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University of Cambridge*

INTRODUCTION

THE English family whose growth is here described consisted of 5 brothers and 5 sisters who were born between 1864 and 1884. Their father measured their height at each birthday, from the first onwards, by standing them with their backs to a board, without shoes, and placing a ruler on their heads, parallel to the floor. The height was then marked on the board at the appropriate level, each child having a separate column. The board also carries measurements of the son of the eldest brother at ages from three to 20 years. These measurements are the material for the present study and are presented for two reasons: first, that longitudinal growth studies, even of one dimension, on so many siblings, are unusual and afford material for the study of genetic variability and of sex differences in growth; and secondly, that such observations are rare in the period under consideration, and are of interest in relation to secular changes in rate of growth.

The family was a well-to-do upper middle class one, and the environment in which the children grew up was as good as was available at that time. All the 5 boys went to English public (private) schools, while the girls were educated at home.

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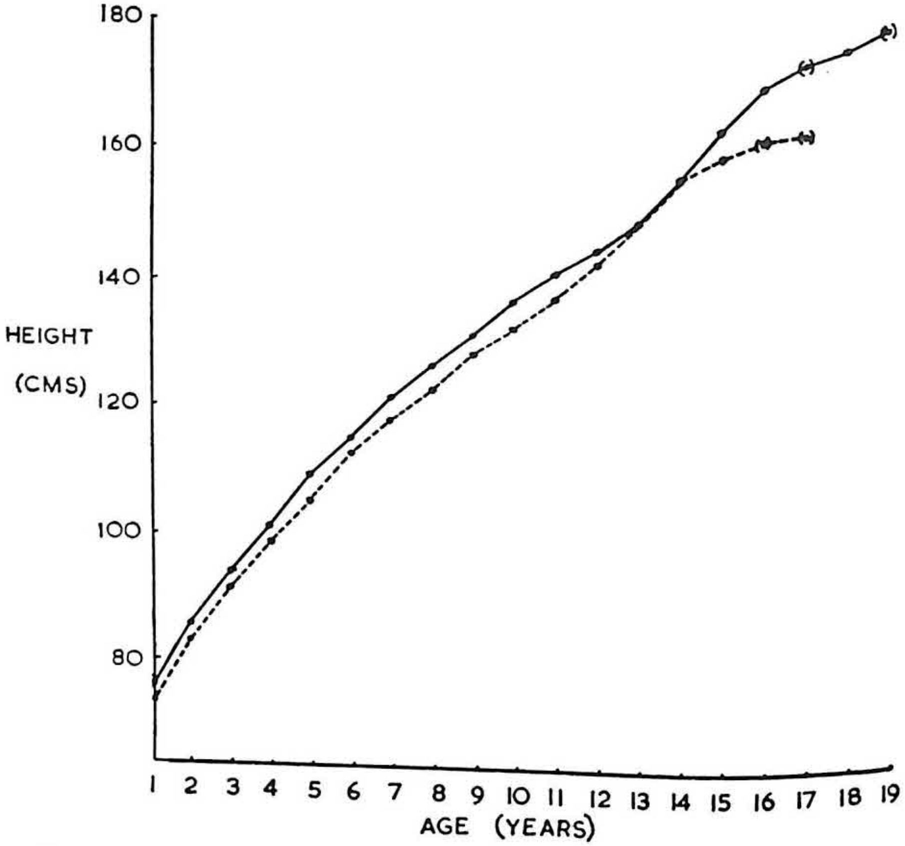

FIG. 1. MEAN GROWTH CURVES FOR MALES (CONTINUOUS LINE) AND FEMALES (DOTTED LINE). THE POINTS IN BRACKETS ARE THOSE FOR WHICH MEASUREMENTS OF ALL 5 BROTHERS OR SISTERS WERE NOT AVAILABLE.

TABLE 2

Total increase in height in cm at each year over 9 (females) and 11 (males)

YEAR	1st	2nd	3rd	4th	5th	6th	7th
Increase (females)	3.9	8.6	14.2	20.5	26.7	29.8	32.0
Increase (males)	3.7	8.0	14.0	21.4	27.8	31.2	33.2

more prolonged growth period (from the age of 9 to 11) of the males, about 2.5 cm were due to greater size of the male before adolescence and about 2.5 cm to greater growth of the males during and after adolescence.

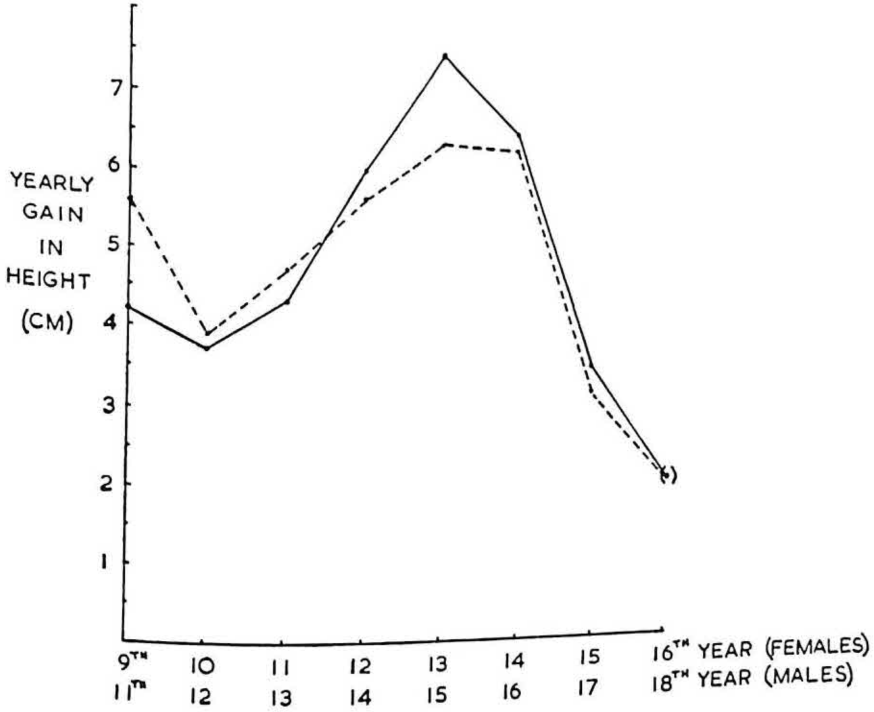

Fig. 2. Yearly Increments of Height During the Adolescent Period of Males (Continuous Line) and Females (Dotted Line) Superimposed by Eliminating the Difference of two Years in Age of Onset of Adolescence.

Evidence from the curves of mean growth for a juvenile (mid-growth) spurt is indecisive; there is a suggestion of a slight temporary quickening of growth at 7-8 years.

Table 3 gives the yearly increments of height of each of the 10 siblings. Males and females will be considered separately. Up to 11 years there was little difference in size between the brothers. The mean range of height was 4.0 cm (1.5-5.4 cm), whereas after 11 was 6.4 cm (4.0-9.6 cm) owing to the variations in time of onset of adolescence. M3 tended to be taller than his brothers at all ages.

There is a suggestion that greater height before adolescence (M3 and M5) was associated with a late onset of adolescence.

TABLE 3

Yearly increments in height, in centimeters

YEAR	M1	M2	M3	M4	M5	F1	F2	F3	F4	F5	S1	MEAN M1-5	MEAN F1-5
2nd	10.9	11.7	10.0	12.7	10.2	9.1	11.4	8.1	10.1	10.5		11.1	9.8
3rd	8.6	7.1	9.3	9.4	7.3	8.7	8.4	10.1	7.6	8.8		8.3	8.7
4th	5.1	9.4	6.6	6.6	8.1	7.1	6.1	6.1	8.5	7.7	10.4	7.2	7.1
5th	10.0	7.4	7.9	7.1	6.6	6.3	6.8	7.4	6.3	5.8	14.5	7.8	6.5
6th	5.1	4.8	6.3	6.6	6.1	5.6	7.6	6.1	8.9	7.0		5.8	7.1
7th	5.7	7.1	4.4	8.6	4.9	6.4	5.6	6.3	4.6	3.9	6.4	6.1	5.3
8th	4.9	4.4	6.8	4.4	5.1	5.6	3.6	3.6	6.8	3.6	6.3	5.1	4.7
9th	4.6	6.0	5.1	4.6	4.7	5.6	8.4	4.1	4.1	5.9	3.8	5.0	5.6
10th	5.3	5.9	4.4	6.1	4.6	2.5	3.0	5.5	4.0	4.5	4.6	5.3	3.9
11th	5.1	4.8	4.0	2.7	4.4	6.3	4.6	4.1	4.1	4.6	4.1	4.2	4.7
12th	4.8	3.3	4.1	3.6	3.0	7.6	4.3	4.1	6.1	5.8	6.8	3.7	5.6
13th	3.6	5.6	4.8	3.3	4.1	5.4	5.9	5.0	6.9	8.1	5.6	4.3	6.3
14th	7.6	6.0	4.8	5.8	5.6	5.6	6.8	7.7	5.3	5.9	7.6	6.0	6.2
15th	9.2	8.5	5.6	6.6	7.1	2.5	4.6	2.8	2.8	2.8	4.8	7.4	3.1
16th	5.7	4.2	9.7	5.4	7.3	1.0	3.0	2.0	1.1	1.8	2.8	6.4	(2.0)
17th	2.4	3.9	5.8	2.0	4.6		3.9	3.8	1.5		2.0	3.4	
18th	1.5		3.0	1.7	2.0				0.7		1.9	(2.0)	
19th	1.5	1.0	0.5	1.1							0.5		
20th													

Between one and 9 years the mean height range was much greater for the females than for the males (7.1 cm). This was largely due to F4, who after the age of 5 was consistently much larger than her sisters and to F1 who was always rather small.

TABLE 4

Years of commencement and of peak velocity of the adolescent growth spurt, and increment of height in the year of peak velocity

BROTHER	YEAR OF COMMENCEMENT	YEAR OF MAXIMUM GROWTH	INCREMENT (CM) IN YEAR OF MAXIMUM GROWTH
M1	14th	15th	9.2
M2	14th	15th	8.5
M3	15th	16th	9.7
M4	14th	15th	6.6
M5	14th	15th-16th	7.3

TABLE 5

Years of commencement and of peak velocity of the adolescent growth spurt, and increment of height in the year of peak velocity

SISTER	YEAR OF COMMENCEMENT	YEAR OF MAXIMUM GROWTH	INCREMENT (CM) IN YEAR OF MAXIMUM GROWTH
F1	11th	12th	7.6
F2	13th	14th	6.8
F3	13th	14th	7.7
F4	12th	13th	6.9
F5	12th	13th	8.1

The time of onset of the adolescent spurt varied by two years in the sisters, but the age of peak growth did not seem to be correlated with size before adolescence as it was in the brothers.

Comparison of the growth curves of F2 and F4 at adolescence is striking. Although there was a difference of a year in time of onset of adolescence, if the two curves are superimposed so that the peak velocities of growth coincide (see figure 3) they appear almost identical in form. Clearly the rate of growth at adolescence was the same for both sisters, although the age of onset differed by a whole year.

In the males the probable order of adult height was as follows: M3, M1, M2, M5, M4. There is no correlation between time of onset of adolescence and adult size, but there may be a correlation between maximum velocity of growth (order M3, M1, M2, M5, M4) and adult height.

In the females the probable order of adult height was as follows: F4, F2, F5, F3, F1. Again there is no correlation between time of onset of adolescence and adult height, nor is there here a relationship between maximum velocity of growth and adult height.

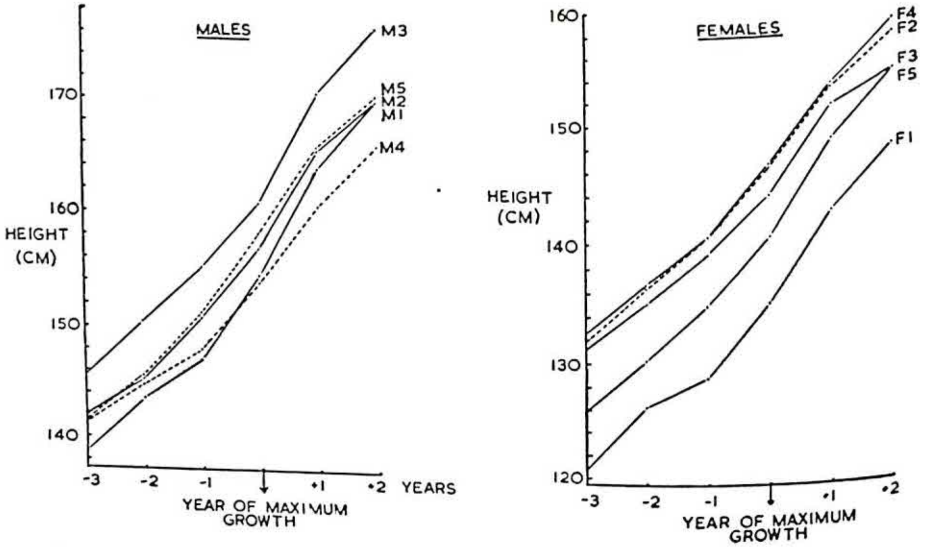

FIG. 3. GROWTH CURVES OF THE 5 BROTHERS (LEFT) AND 5 SISTERS (RIGHT) SUPERIMPOSED BY MAKING THE AGE AT WHICH MAXIMUM GROWTH TOOK PLACE THE SAME FOR ALL, REGARDLESS OF ACTUAL AGE. THERE IS LITTLE CHANGE IN ORDER OF HEIGHT DURING ADOLESCENCE.

S1, the son of M1, was born in 1925, nearly 60 years after his father. His height at ages 3-20 is shown in table 1 and the yearly increments in table 3. The mother's family was of a similar average stature to the father's. Although the adult difference in size between S1 and M1 was only 2.5 cm, S1 was taller than M1 at all ages (table 6) and taller than

TABLE 6

Difference in height between S1 and M1 at ages 3-18

AGE	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Height difference (cm)	2.1	7.4	—	6.8	7.5	8.9	8.1	7.4	6.4	8.4	10.4	10.4	6.0	3.1	2.7	3.1

any of his uncles up to the age of 16. The adolescent spurt began in the 12th year and reached its peak velocity in the 14th, a year earlier than in any of the previous generation. The relation of difference in height between M1 and S1 for ages between three and 14 has been calculated by the method of least squares with the following result. Height difference $= 0.28 \times$ age $+ 3.94$. Table 7 gives the calculated values for the height difference at ages from 3 to 14 and also these values expressed as percentages of the height of M1 at each age.

TABLE 7

Calculated height differences between M1 and S1 at ages 3-14

AGE	3	4	5	6	7	8	9	10	11	12	13	14
Calculated height difference (cm)	4.8	5.1	5.3	5.6	5.9	6.2	6.5	6.7	7.0	7.3	7.6	7.9
Height difference as % of height of M1	5.2	5.2	4.9	4.9	4.9	4.9	5.0	5.0	5.1	5.1	5.2	5.2

At all ages from 3 to 14, S1 was about 5% taller than M1 and since he attained adolescence about a year earlier it appears that this difference was due to more rapid growth rather than to absolute greater size. During and after the adolescent spurt the difference in size was only about 2.5 cm at comparable stages of maturity, which is considerably less than the difference before adolescence and hardly significant.

DISCUSSION

Comparison of the growth of the two sexes

Since the age of onset of adolescence and of peak growth velocity varied within each sex, the mean growth curves tend to blur the sharp changes in growth rate which occurred at those times. However, they have the advantage that they tend to cancel out both small measurement errors which deform the individual curves and also any genetic variability which is equally distributed within the two sexes. Thus they enable a better general comparison to be made of the growth of the two sexes than is obtained by comparing the growth of two individuals. They show that on average the females started their adolescent spurt two years before the males, as at the present day (Tanner, '55), and that the actual duration and form of the spurt was strikingly similar in the two sexes. To demonstrate this fact the mean increments of height for the year of maximum growth velocity and for the two preceding and three following years have been expressed in table 8 as a percentage of height.

This indicates that the specific growth-rate during the adolescent spurt (that is, growth expressed as self-multiplication of actual height) was virtually identical in the two sexes. The actual yearly height-increments at adolescence were, however, greater in the males since their absolute stature was greater at a comparable stage during the adolescent spurt. The only striking difference between the sexes was that growth in the females was more sustained in the second and third years after the year of peak growth velocity.

TABLE 8

Mean yearly height increments at adolescence expressed as percentage of height at beginning of year

(YEARS)	— 2	— 1	YEAR OF PEAK GROWTH VELOCITY	+ 1	+ 2	+ 3
Males	3.13	4.29	5.24	3.14	1.33	0.73
Females	2.94	4.27	5.21	3.18	2.10	1.40

Order of individual size during the adolescent growth spurt

If the order of size of the members of each sex is compared in the year of maximum growth velocity and in the years preceding and following this (figure 3), it is found to be remarkably constant and corresponds closely to order of size at completion of growth, which was therefore related both to the size at which the growth spurt began and also to the size at which maximum growth was occurring. Those who were larger at any particular phase of the growth spurt were larger as adults.

S1 was consistently larger than M1 and smaller than M3 and M5 at a comparable stage of maturity, and this corresponded to his actual adult height in relation to M3, M5 and M1. Despite quicker maturation and an earlier onset of adolescence, his size during adolescence and in adult life was well within the size range of the previous generation.

Relation of size before adolescence to onset of adolescent growth spurt.

It has long been known (Baldwin '21, quoted by Tanner, '55; Boas '32) that children who are tall before puberty begin adolescence earlier than those who are short. This is only partly true because in a large group of children the tallest before adolescence are usually those who are maturing most quickly and who have the greatest skeletal age; for Bayley ('46) and Simmons and Greulich ('43) have shown that time of onset of adolescence is more closely related to skeletal than to chronological age; and size at maturity can be more readily predicted from size at a given skeletal age than from size at a comparable chronological age.

This however ignores the influence of physique on rate of development, and it has been shown by Dupertuis and Michael ('53) that ectomorphic boys are taller than mesomorphic boys before adolescence at all ages over 4 (though this finding is not statistically proven), and that they attain adolescence about a year later. Thus genetically tall individuals may mature later than genetically short ones, if their tallness is associated with greater ectomorphy.

This point may be illustrated in the present study. M3 and M5 were the tallest of the brothers during the adolescent spurt. M3 was consistently the tallest before adolescence, and M5 was the second tallest up to the age of 6. Both of them attained adolescence later than their brothers. Greater height seems to have been due in these two brothers not to greater rapidity of development but to a genetic height difference from their brothers, which could possibly have been associated with greater ectomorphy.

Among the sisters F4 and F5 reached adolescence a year earlier than F2 and F3, while F1 did so a year earlier still. F1 differed from her sisters when adult, being smaller and less mentally acute, and it is possible that a thyroid abnormality in early childhood may have accounted for her early maturity and small adult size. The growth at adolescence of F2 and F4 was strikingly similar, both in rates of acceleration and deceleration of growth and in actual size at each phase. This suggests that a genetic similarity in height was associated with a genetic difference in speed of development before adolescence, since it is unlikely that their environments could have differed sufficiently to produce a full year's difference in onset of adolescence. Thus in the sisters, unlike the brothers, greater height before adolescence (in F4 and F5) was associated with more rapid development and with earlier adolescence. In the family as a whole there appear to be at least two independent hereditary factors affecting height at a given age, one determining height at a given stage of maturity and the other rapidity of growth and age at adolescence.

Environmental influences—secular trend towards more rapid growth

The increased rapidity of growth and maturation of S1 may reflect the secular changes in growth known to have taken place in the last 100 years in many countries; in England and Scotland (Weir '52; Clements '53), Sweden (Broman, Dahlberg and Lichtenstein '42; Abramson and Ernest '54), the U. S. A. (Meredith '41), Canada (Meredith and Meredith '44; Hopkins '47) and elsewhere. As Weir has pointed out these changes are due to an acceleration of growth prior to adolescence, giving a greater size at any given chronological age, although size in relation to skeletal age may not be increased.

Although no conclusions can be drawn from the growth of S1 in the absence of other data for the second generation, the fact that adolescence started a year earlier than for any of the previous generation of males, but that the height was no greater at a comparable stage of adolescence, is certainly suggestive of a secular change of the type which Weir ('52) describes.

SUMMARY

1. Variations of growth in height have been studied in a family of ten siblings born between 1864 and 1884.
2. The specific growth-rate during the adolescent spurt of growth was the same for both sexes. Greater increase in size of the males at this time was due to greater absolute size.
3. Two-thirds of the greater size of the males was due to a longer period of growth. The remaining third was due partly to greater absolute size before adolescence and partly to greater size increase at adolescence.
4. There was no straightforward relationship between height and time of onset of the adolescent spurt, since height at a given age was governed by two independent hereditary factors, one controlling rate of maturation and the other the height at any given stage at maturity.
5. Since the rate of growth during and after adolescence was similar for all members of the family, adult size was closely related to size at the beginning and at the peak of the adolescent spurt of growth.
6. A son of one of the brothers, born nearly 60 years after his father,

grew 5% faster before adolescence and matured a year earlier than his father. His size and growth-rate during adolescence and his adult height did not differ significantly from those of the previous generation.

7. No clear evidence was found for the existence of a juvenile growth spurt.

ACKNOWLEDGMENTS

I should like to express my gratitude to Professor J. D. Boyd of the Anatomy School, University of Cambridge, and to Dr. J. M. Tanner of the Institute of Child Health, University of London, for reading the manuscript and for giving their criticism and advice.

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However, this term was not precise because it contained the effect of sampling errors. Therefore, an approximate test was used (described in Appendix) which enabled the deletion of this effect to make the estimate of appropriate error more precise. With this adjustment, the inter-anthropometrist effect appeared in knee height, buttock-knee length, buttock-leg length, anterior arm reach, and maximum arm reach. This effect contributed the largest proportion of total variance in the last named feature (10%).

TABLE 1

Estimates of components of variance and the % of total variance contributed by each

MORPHOLOGICAL FEATURE	INTRA-SUBJECT VARIANCE	% OF TOTAL	INTER-SUBJECT VARIANCE	% OF TOTAL	INTRA-ANTHROP. VARIANCE	% OF TOTAL
Buttock-leg Length	6.45	19.5	26.65	80.5	*	
Height	.13	.3	37.07	99.7	*	
Weight	1.05	.30	352.66	99.68	.07	.02
Knee Height	.22	3.5	6.00	96.5	*	
Buttock-knee Length	.44	6.6	6.20	93.4	*	
Elbow-elbow Breadth	.88	9.2	8.67	90.8	*	
Bideltoid Diameter	.32	9.0	3.24	91.0	*	
Max. Arm Reach	1.92	10.8	15.77	88.2	.18	1.0
Span	1.76	4.4	40.67	95.6	*	
Ant. Arm Reach	2.30	17.4	10.68	80.8	.23	1.8
Sitting Height	.57	5.9	9.09	94.1	*	

NOTE—Inter-Anthropometrist Variance was non-existent and is not included in the table.

* Not significant; therefore, considered non-existent.

According to table 3 the morphological features which are most reliable are height, weight, span, and sitting height; the least reliable are buttock-leg length, maximum arm reach, and anterior arm reach. The great variability seen in the arm reaches is to be expected inasmuch

TABLE 2

Estimates of components of variance and the % of total variance contributed by each using the approximate test for evaluation of the inter-anthropometrist effect

MORPHOLOGICAL FEATURE	INTRA-SUBJECT VARIANCE	% OF TOTAL	INTER-SUBJECT VARIANCE	% OF TOTAL	INTER-ANTHROP. VARIANCE	% OF TOTAL	INTRA-ANTHROP. VARIANCE	% OF TOTAL
Buttock-leg Length	6.45	19.4	26.18	78.6	.67	2.0	*	
Height			no change					
Weight			no change					
Knee Height	.22	3.5	5.73	90.2	.40	6.3	*	
Buttock-knee Length	.44	6.5	5.95	88.0	.37	5.5	*	
Elbow-elbow Breadth			no change					
Bideltoid Diameter			no change					
Max. Arm Reach	1.92	10.4	14.45	78.6	1.83	10.0	.18	1.0
Span			no change					
Ant. Arm Reach	2.30	17.5	10.09	76.4	.58	4.4	.23	1.7
Sitting Height			no change					

* Not significant; therefore, considered non-existent.

TABLE 3

Percentage of total variance contributed by each component of variance

MORPHOLOGICAL FEATURE	RELIABLE VARIANCE		UNRELIABLE VARIANCE	
	INTER-SUBJECT	INTRA-SUBJECT	INTER-ANTHROP.	INTRA-ANTHROP.
Height	99.7	.3		.02
Weight	99.68	.30		
Span	95.6	4.4		
Sitting Height	94.1	5.9		
Bideltoid Diameter	91.0	9.0		
Elbow-elbow Breadth	90.8	9.2		
Knee Height	90.2	3.5	6.3	
Buttock-knee Length	88.0	6.5	5.5	
Buttock-leg Length	78.6	19.4	2.0	
Maximum Arm Reach	78.6	10.4	10.0	1.0
Anterior Arm Reach	76.4	17.5	4.4	1.7

as movement of the shoulder joint varies in three dimensions. Any measurement involving this joint will tend to offer a sizeable amount of unreliable variance through both intra-subject and inter-anthropometrist sources of variation.

A procedure by which to overcome the unreliability indicated in these analyses would be to obtain more than one sample of each measurement for each morphological feature, with some time interval between measurements, thereby increasing the reliability of the estimate. Such a procedure would be of special value in the conducting of anthropometric studies which are concerned with the solution of practical problems such as fitting various personnel with clothing or other equipment. However, this procedure would result in great expense. A compromise solution which would seem feasible would be to take only one measurement on those morphological features which have been indicated to be most reliable and to obtain two or more for the more variable ones.

ACKNOWLEDGMENT

The authors wish to express their appreciation to Mr. James Young of the Air Crew Equipment Laboratory for his valuable assistance as an examiner in the prosecution of the experiment.

APPENDIX

The analysis of variance model used in deriving estimates of the components of variance for the 11 morphological features is described by Harter and Lum ('55) and by Greenwood ('56). The paradigm is shown in table 4. There were three random effects, Subjects (which "nested" within Anthropometrists), Anthropometrists, and Trials, which represented Inter-Subject, Inter-Anthropometrist, and Intra-Anthropometrist sources of variation, respectively. The Subjects X Trials Interaction Within Anthropometrists is used to represent Intra-Subject variance because the entire experiment could not be replicated to give an estimate of experimental error. However, the very nature of the experiment seems to indicate that this interaction is non-existent. Therefore, σ^2ac can be deleted from the Expected Value of Mean Square column in table 4, leaving only σ^2e; σ^2e contains intra-subject variations and any other uncontrolled aspects in the experiment. However, inasmuch as the other sources of variation are considered by the Trials and by the Anthropometrist effect, σ^2e should contain only intra-subject variations.

It is obvious from the table which effects are used as the appropriate error term for each test of significance. However, there is no test of the Anthropometrist effect unless the Trials X Anthropometrist Interaction effect is non-significant. In all analyses this effect was non-existent. Inasmuch as the question of the pooling of non-significant interactions with the error term is far from settled at the present time, a "never pool procedure" was adhered to in making tests of significance. However, the Subjects X Trials Interaction Within Anthropometrists was used whenever it contained the same terms as the effect which was the appropriate error term, because it was based on greater degrees of freedom and, thus, it was a more reliable estimate. After all effects had been shown to be present or non-existent by tests of significance, all effects containing the same terms were pooled and estimates of the various components of variance, and the percentage of total variation contributed by each of these was obtained.

TABLE 4

Components of variance paradigm

SOURCE OF VARIATION	df	EXPECTED VALUE OF MEAN SQUARE
Anthropometrist (b)	2	$\sigma^2e + 14\sigma^2ab + 3\sigma^2c + 42\sigma^2b + \sigma^2ac$
Subjects Within Anthropometrists (c/b)	39	$\sigma^2e + 3\sigma^2c + \sigma^2ac$
Trials (a)	2	$\sigma^2e + 14\sigma^2ab + 42\sigma^2a + \sigma^2ac$
Trials X Anthropometrist (ab)	4	$\sigma^2e + 14\sigma^2ab + \sigma^2ac$
Subjects X Trials Within Anthropometrists (ac/b)	78	$\sigma^2e + \sigma^2ac$

σ^2a = Intra-Anthropometrist Variance; σ^2b = Inter-Anthropometrist Variance; σ^2c = Inter-Subject Variance; σ^2e = Intra-Subject Variance.

In all 11 analyses the Anthropometrist effect was non-significant when tested by the Subjects Within Anthropometrist effect. (Trials by Anthropometrist was non-existent in all 11 analyses). The Anthropometrist effect contains sampling errors and is less precise than a within-subjects effect. Therefore, an approximate test of the Anthropometrist effect was made by deleting the effects of sampling errors (σ^2c) by the following F test:

> Anthropometrist mean square — Subjects Within Anthropometrists mean square + Subjects X Trials Within Anthropometrists mean square divided by Subjects by Trials Within Anthropometrists mean square.

In terms of the Expected Value of Mean Square, after the nonsignificant components have been deleted, this F test is as follows:

$$\frac{\sigma^2 e + 3\sigma^2 c + 42\sigma^2 b - (\sigma^2 e + 3\sigma^2 c) + \sigma^2 e}{\sigma^2 e} \text{ which reduces to } \frac{\sigma^2 e + 42\sigma^2 b}{\sigma^2 e}.$$

When this approximate test is used the Inter-Anthropometrist effect was significant in 5 of the morphological features. However, it must be emphasized that this test, even though theoretically sound in principle, must be considered as an approximation inasmuch as an appropriate test had been provided by the mathematical model. An experimental design in which all effects are within-subjects effects would overcome this difficulty and give a precise error term for all effects, e. g., a treatments X subjects design. This design would require that the anthropometrists take two or more measurements on every subject. This possibility had to be excluded in the present study because the subjects were only available for a few sessions.

To illustrate the use of this statistical design, tables 5, 6, and 7 present the data for the analysis of maximum arm reach measurements. This analysis is chosen because each of the 4 components of variance contribute to the total variation for this feature.

TABLE 5

Analysis of variance of maximum arm reach measurements

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	P
Anthropometrist	2	244.02	122.01	2.70	$>.05$
Subjects Within Anthropometrists	39	1765.32	45.26	23.70	$<.001$
Trials	2	18.90	9.45	4.95	$<.01$
Trials X Anthropometrists	4	8.82	2.21	1.16	$>.05$
Subjects X Anthropometrists Within Anthropometrists	78	148.92	1.91		
TOTAL	125	2185.98			

TABLE 6

Approximate test of anthropometrist effect

$$F = \frac{122.01 - 45.26 + 1.91}{1.91} = \frac{78.66}{1.91} = 41.18 \quad P < .001$$

TABLE 7

Estimates of components of variance for maximum arm reach measurements

SOURCE OF VARIATION	MEAN SQUARE	COMPONENTS
Anthropometrist (*b*)	122.01	$\sigma^2 e + 3\sigma^2 c + 42\sigma^2 b$
Subjects Within Anthropometrists (*c*)	45.26	$\sigma^2 e + 3\sigma^2 c$
Trials (*a*)	9.45	$\sigma^2 e + 42\sigma^2 a$
Subjects X Trials Within Anthropometrists (*e*)	1.92	$\sigma^2 e$

			Estimate of variance	*% of total*
$\sigma^2 e =$			1.92	10.4
$\sigma^2 a =$	$\frac{9.45 - 1.92}{42}$	=	.18	1.0
$\sigma^2 c =$	$\frac{45.26 - 1.92}{3}$	=	14.45	78.6
$\sigma^2 b =$	$\frac{122.01 - 45.26}{42}$	=	1.83	10.0
			18.38	100.0

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THE SUCKING BEHAVIOR IN PREMATURE INFANTS *

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INTRODUCTION

ONE of the most vital patterns of behavior in infancy is the sucking reflex. This phenomenon, though commonly observed and simple in manifestation, is perhaps one of the least understood, and has to some extent been neglected experimentally. Perusal of the medical literature reveals a dearth of information on the subject.

Sucking represents the interaction of an inborn drive and a conditioned reflex. Although it is unlearned behavior, it can be intensified, strengthened, or changed in accordance with learning experiences.

Reference to the literature reveals two principal theoretical points of orientation from which to examine the sucking phenomenon: namely, (1) the Learning theory and (2) the Psychoanalytic theory.

According to the Learning theory, sucking is regarded as a stimulus-response mechanism with hunger as the biological drive determinant. For example, when the infant is hungry and a nipple is placed in his mouth ("stimulus"), he makes spontaneous sucking movements ("response") in reaction to the applied stimulus. This is an unlearned pattern of behavior. The act of sucking, however, brings gratification and the response is rewarded and therefore strengthened ("conditioned"). As a result, the behavior pattern becomes more fixed, and increases in intensity. It has been "reinforced."

The Psychoanalytic theory holds that sucking is an instinctual form of behavior, prompted by libidinal as well as physiological needs. According to this view, infants with less opportunity to suck might be expected to show signs of emotional disturbance.

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Davis and his associates ('48) studied the effects of feeding 20 infants by cup, 20 by bottle, and 20 by breast during the first 10 days of life. During the period of study, the breast-fed infants developed a stronger sucking reflex than the other two groups, but surprisingly there were fewer signs of frustration or emotional disturbance in the cup-fed babies than in those fed by breast. Moreover, the bottle-fed infants did not, as would be expected from the Learning theory, show a sucking behavior similar to that of the breast-fed group.

A number of reports are available which describe oral behavior of infants or young animals (Brazelton, '56), but none could be located which sought to correlate the sucking reflex with the maturity of the infant. As in the case of other vital functions, the sucking reflex is established prenatally (Hooker, '42) and develops in complexity as the fetus approaches maturity. There are no clear-cut data indicating when, in prenatal life, the full potential for the sucking reflex should be reached, or in more general terms, when a plateau or leveling-off period in postnatal life occurs.

Gesell ('45) describes the behavioral characteristics of the fetal-infant (premature) whom he defines as, ". . . an individual born and living in the fetal period." He states that, "In previous studies we have followed the after-career of healthy premature infants. These studies have shown that the fundamental rates and patterns of development are little disturbed by displacements of birth. The infant remains true to his fetality—even though born before his time. He cleaves close to the inherent sequence of maturation."

Since the mechanism of sucking is developed during prenatal life, its presence, absence, and degree of intensity after birth should give some indication of gestation time. It was postulated that the maturation of the sucking behavior might follow a course parallel with or approximating that of other aspects of growth and development. If so, then the premature infant from a 7.5 month gestation period might be expected to reach his peak in sucking efficiency or maturity at approximately 1.5 months (45 days) postnatally, assuming that the reflex is fully developed in the full-term infant.

METHODS AND MATERIALS

The present study was designed to measure the fluid intake of premature infants within a prescribed time interval at regular periods during the infant's stay in the hospital, as an index of sucking power. It may be divided conveniently into three phases:

Maximum Rate of Water Intake During First Three Days of Life

First, an attempt was made to determine the sucking efficiency of the premature infant shortly after birth and throughout the first 72 hours postnatally. For this purpose, 52 Negro female [1] premature infants were fed sterile water daily at the time of the regular 9 a. m. feeding, when the hunger drive is expected to be very near maximal.

For the purpose of test-feeding, a one-ounce (30 cc) graduated bottle was used with soft rubber nipple. The opening in the nipple was 2 millimeters in length and was prepared by means of a small chisel-like blade originally used for scratch testing in allergy.

When the nipple was inserted into the mouth and the infant began sucking, a stop-clock was set. The amount of water taken in a two-minute interval was ascertained and recorded. If the infant consumed the full amount in less than the stated interval, the number of seconds required was recorded.

Individual differences in continuity of sucking were observed, but no allowance was made for pauses within the two-minute interval. In the rare instance in which a baby seemed to be sucking so fast as to choke, the nipple was withdrawn and the experiment discontinued for that day. If an infant became ill, she was dropped from the project. The sucking rate (cc of fluid consumed per second) is determined by using the simple formula,

$$\text{Rate} = \frac{\text{Volume consumed (cc)}}{\text{Time (seconds)}}.$$

It was noted that in a 30 cc bottle, approximately 0.5 cc clings to the container and cannot be removed by sucking. Hence, when the infant empties the full bottle, the intake is recorded as 29.5 cc.

Only the maximum rate of sucking efficiency is considered in this study, and this was determined simply by ascertaining the day when the infant consumed the most liquid in the least time.

Maximum Sucking Efficiency of the Premature Infant and the Normal Newborn During the First Three Days of Formula Feeding

It was deemed advisable to study the sucking behavior of a small sample of full-term infants as a frame of reference, or standard, for evaluating the observations made on premature infants. For this purpose 32 normal newborn Negro infants were tested during the first three days of life in a manner identical with that described above, except that a standard evaporated milk formula (20 calories per ounce) was used instead of sterile water. The results obtained were compared with those which followed the performance of premature infants who were fed the premature nursery formula (24 calories per ounce) from the fourth to the sixth day of life.

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[1] Only females were used inasmuch as males were the subjects of another study.

Maximum Sucking Rate Beyond the First Postnatal Week

The premature infant population provided an excellent opportunity to observe the baby for a long period of time in the nursery, as opposed to the full-term infants whose length of stay rarely exceeded 72 hours. This prolonged period of hospitalization thus made it possible to ascertain, more nearly, the ultimate maximum sucking rate. Daily contact was maintained with 52 Negro premature infants, through test-feeding with nursery formula at the 9 a. m. feeding period, as described above, for periods varying from 9 to as long as 57 days.

RESULTS

Maximum Rate of Water Intake During First Three Days of Life

When fed sterile water during the first 72 hours of life, it was observed in general that, of the 49 premature infants tested, the heavier babies demonstrated a stronger and more effective sucking reflex (figure 1). When a Chi-Square test was performed, the association between birth weight and maximum rate of water intake was found to be direct and highly significant (1% level of confidence).

The mean maximum sucking rate for water for the 49 infants during the first three days of life was 0.16 cc per second, while that for formula during the fourth to the sixth day of life was 0.25 cc per second. This, however, does not quite reach the accepted level of statistical significance for a mean difference.

There is a direct association between the maximum water rate and maximum formula rate, indicated both by regression analysis and by Chi-Square. Thus, infants with a high water rate tended to have also a high formula rate. The Phi coefficient of correlation, corrected for dichotomized variables, was 0.7948 for the 49 infants studied.

Early Maximum Sucking Rate in Premature and Full-Term Infants

Maximal sucking rate data, collected during the first week of life, are available on 51 premature and 32 full-term infants. The descriptive data on the full-term infants reveal the wide range of sucking efficiency found in normal newborn babies (0.2-1.2 cc per second) and demontrate, moreover, that the sucking rate of a given full-term infant is more or less independent of his birth weight. Figure 2 reveals the

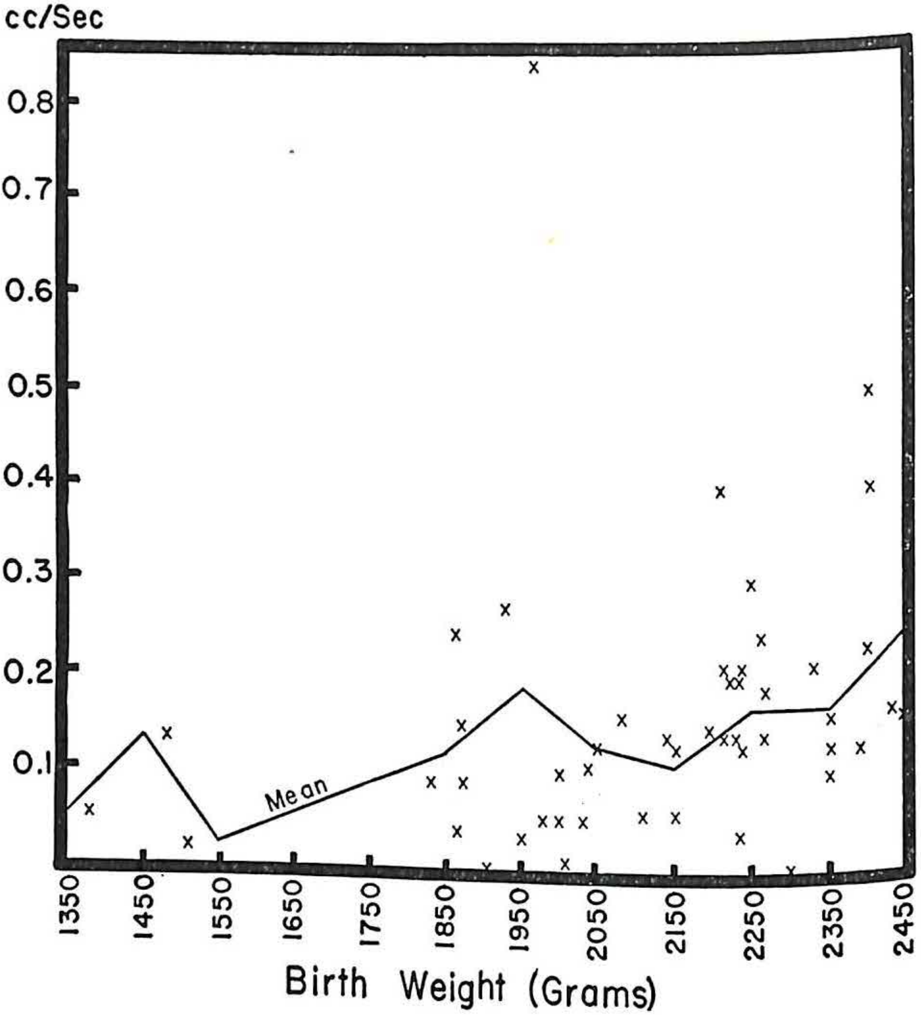

Fig. 1. Maximum Sucking Rate (Sterile Water) of 49 Premature Infants During First Three Days of Life, by Birth Weight.

superior performance of the full-term newborn (0.6 cc per second) during the first three days of formula feedings, as compared with the premature infant (0.24 cc per second). A gross comparison of the maximal sucking efficiency of premature and full-term infants would be expected to show a significant difference in intake during the first week of life. A finer level of discrimination is necessary, however, to determine if a progressive correlation exists between the sucking rate and the infant's weight at birth. Figure 3 reveals the maximum sucking rates of infants, during the first three days of formula feeding, with birth

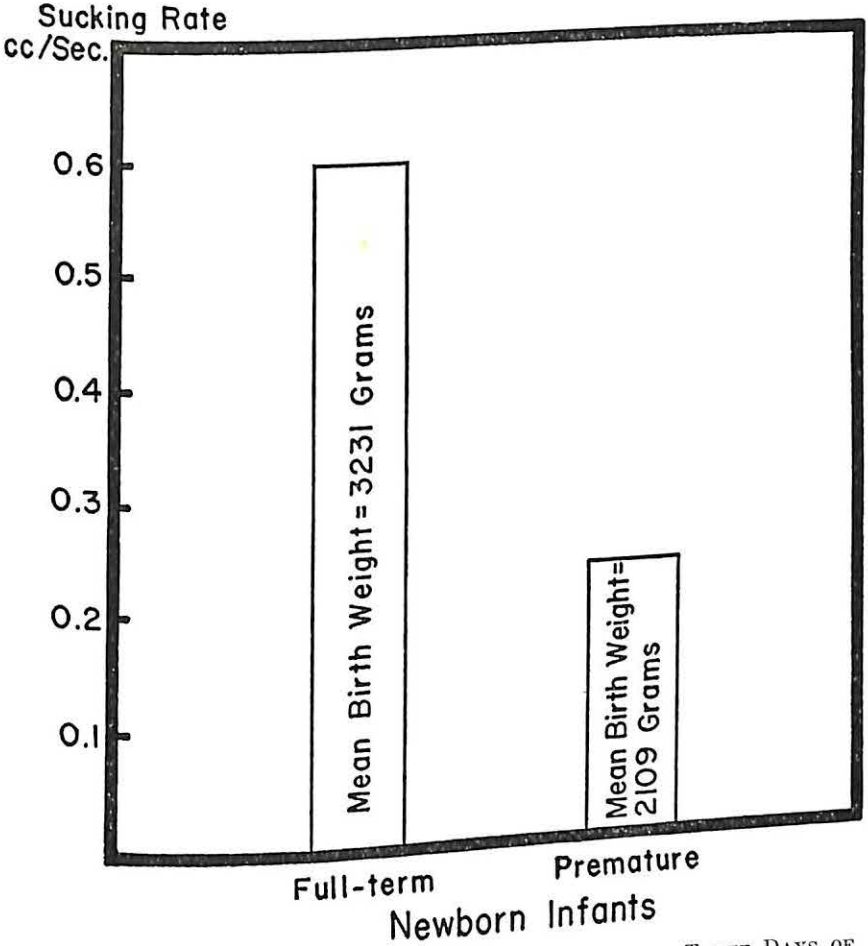

FIG. 2. MAXIMUM SUCKING RATES (MEAN) WITHIN FIRST THREE DAYS OF FORMULA FEEDING (32 FULL-TERM AND 51 PREMATURE INFANTS).

weights ranging from 1380 to 4050 grams. A significant, direct association is shown between the birth weight of premature infants and sucking efficiency. It is observed that, in general, the larger the premature infant at birth the greater his sucking rate. While it is apparent

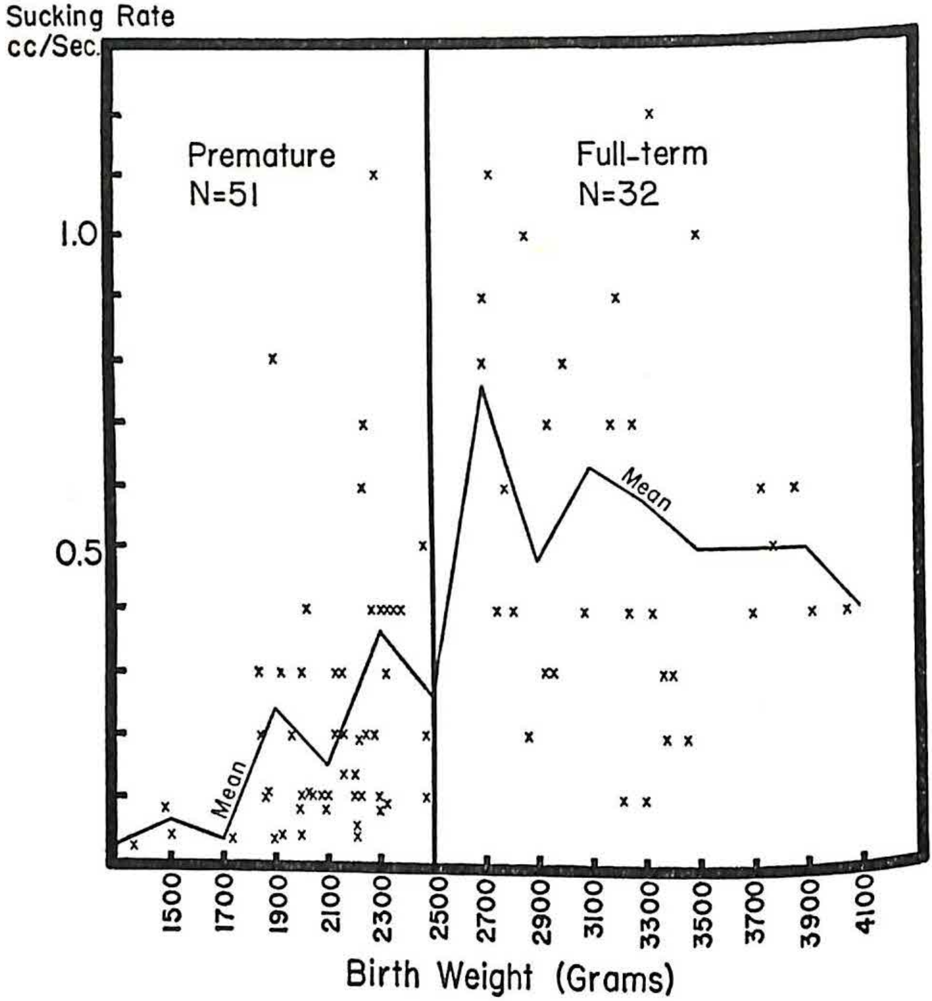

FIG. 3. MAXIMUM SUCKING RATE OF 83 NEWBORN INFANTS DURING FIRST THREE DAYS OF FORMULA FEEDING, BY BIRTH WEIGHT.

that, with respect to position on the birth weight scale, no such progressive association between birth weight and sucking efficiency exists within the full-term group, there is a general tendency toward a plateau or leveling-off of the sucking rate among mature newborn infants, without particular regard to relative size at birth.

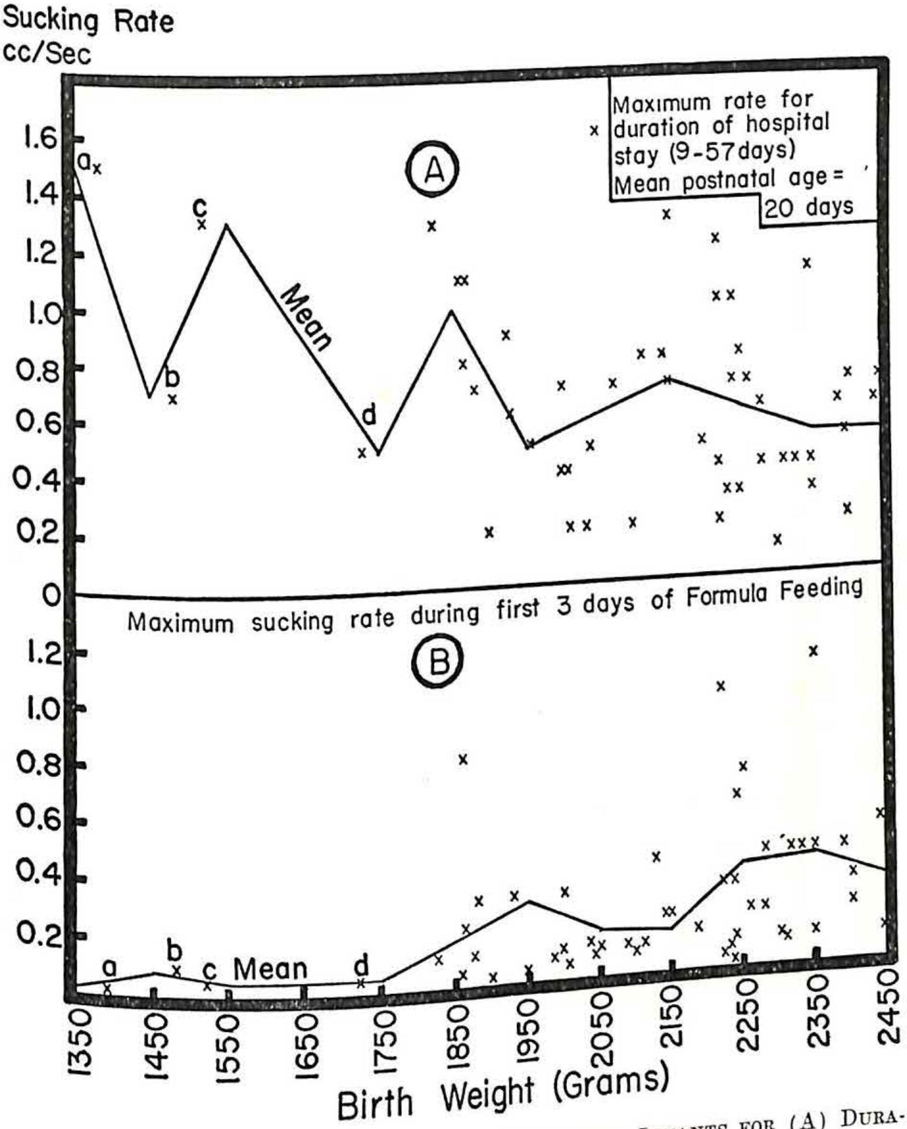

FIG. 4. MAXIMUM SUCKING RATE OF 51 PREMATURE INFANTS FOR (A) DURATION OF HOSPITAL STAY AND (B) FIRST THREE DAYS OF FORMULA FEEDING, BY BIRTH WEIGHT.

Later Maximum Sucking Rate

Because premature babies remained in the nursery for varying periods of time, data on maximum sucking rates are available on 52 infants, ranging in age from 5 to 51 days at the time when maximum sucking efficiency was achieved. The mean postnatal age for the group at maximum rate of intake is 20 days. It was postulated at the outset that in the case of premature infants, a progressive association is likely to exist between size at birth and efficiency of the sucking mechanism as the infant grows older postnatally. In general, this hypothesis seems to be confirmed by data from the present study.

The positive association existing between birth weight and the maximum sucking rate during the first three days of formula feeding (figure 4-B) seems to be obscured later when the same infants (figure 4-A; a, b, c, d) are followed for a longer period of time to ascertain the point at which the highest sucking rate, for the entire period of hospitalization, is achieved. It must, however, be borne in mind that figure 4-A relates birth weight to maximum sucking rate without regard to postnatal age. Moreover, perusal of figure 5 will reveal that infants, a, b, c and d, though smallest by birth weight, are among the oldest [2] postnatally when the maximum sucking rate was achieved.

Figure 6 shows the relationship between the postnatal age of 52 premature infants at the time when the maximum sucking rate was achieved and the age at discharge from the hospital. It will be noted coincidentally that, in most instances, the infant was sent home within three to 5 days after achieving the maximum efficiency in sucking, and that at discharge the babies ranged in age from 9-57 days. Ideally, it would have been desirable to retain all infants in the hospital for a uniform duration of time and to compare the observed maximum sucking rate following identical periods of observation. Inasmuch as the larger babies went home first, the question arises, "Does the maximum sucking rate observed during hospitalization represent the ultimate maximum rate for these infants?" In other words, "Will the rate continue, for a time, to rise following discharge, and then stabilize at a higher level?" This question we cannot answer. However, figures 1 and 3 indicate that sucking operates at a relatively low level of efficiency in the newborn infant (prematures 0.24 cc per second; normal newborns 0.6 cc per second) during the initial phases of feeding. Given time, however, this rate rises with varying degrees of rapidity in the premature infant (figure 4-A). These observations, taken together, lend support to the postulate that the maturation of the sucking mechanism parallels other aspects of growth and development in prematures.

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[2] 38, 35, 46 and 50 days respectively.

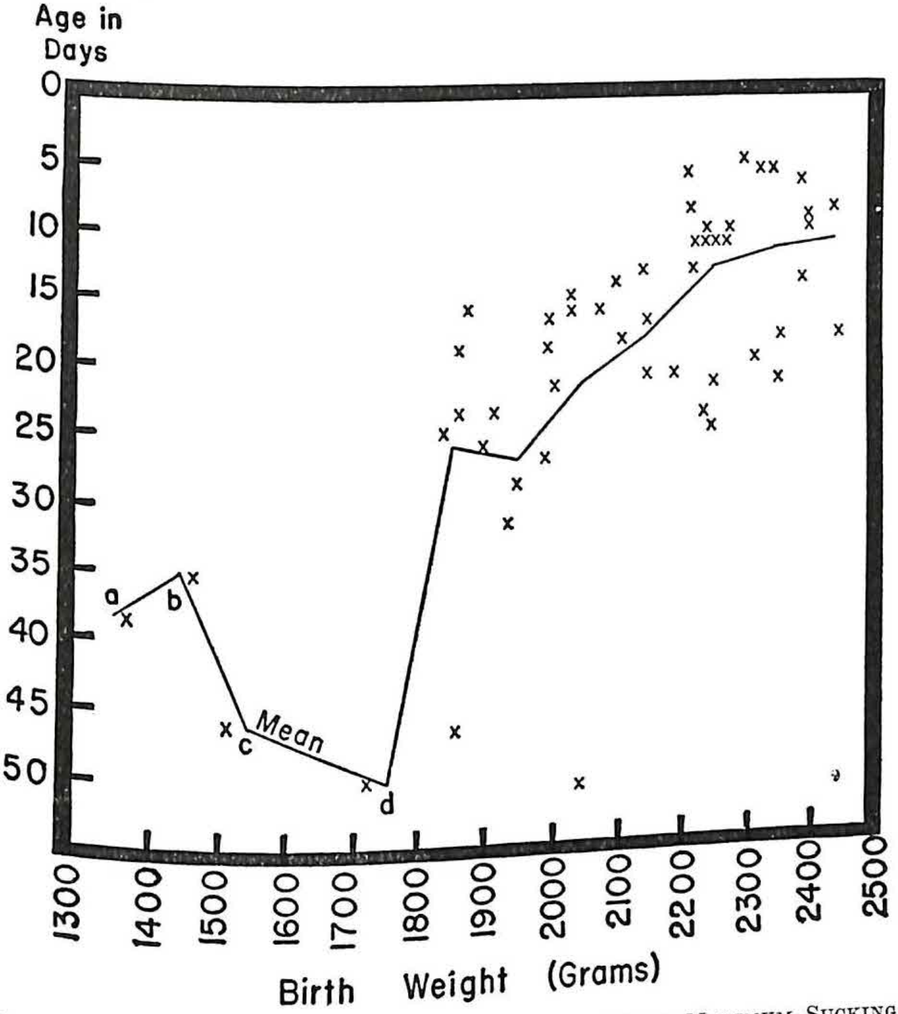

Fig. 5. Postnatal Age of 51 Premature Infants When Maximum Sucking Rate was Achieved, by Birth Weight.

Postnatal Age and Maximum Sucking Rate

Generally speaking, the heavier the premature infant at birth, the younger he was, in terms of postnatal age, when the maximum sucking rate was achieved (figure 5). For example, the average postnatal age at which maximal efficiency in sucking was determined for infants whose birth weight was less than 2000 grams was 30 days, while the corresponding age for those weighing 2000-2500 grams was 15 days.

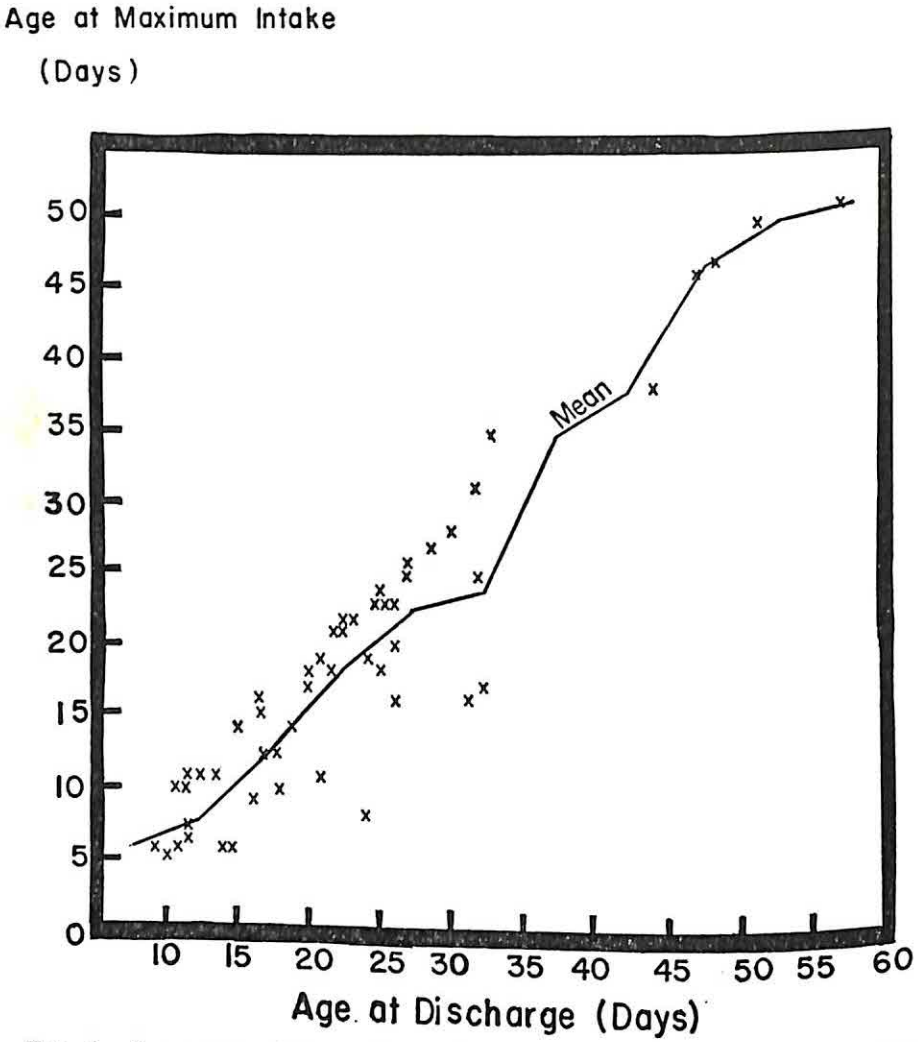

FIG. 6. POSTNATAL AGE IN DAYS WHEN MAXIMUM SUCKING RATE WAS ACHIEVED, BY POSTNATAL AGE IN DAYS AT DISCHARGE FROM HOSPITAL, FOR 52 PREMATURE INFANTS.

A Chi-Square test for independence between postnatal age when the maximal sucking rate was observed and the magnitude of the maximal rate revealed a positive association, significant at the 5% level of confidence (figure 7).

Relationship of Gestational Age to Maximal Sucking Efficiency

Age of gestation[3] was available for 23 of the 52 premature infants studied. Scattergrams prepared for these 23 revealed no significant association between the following:

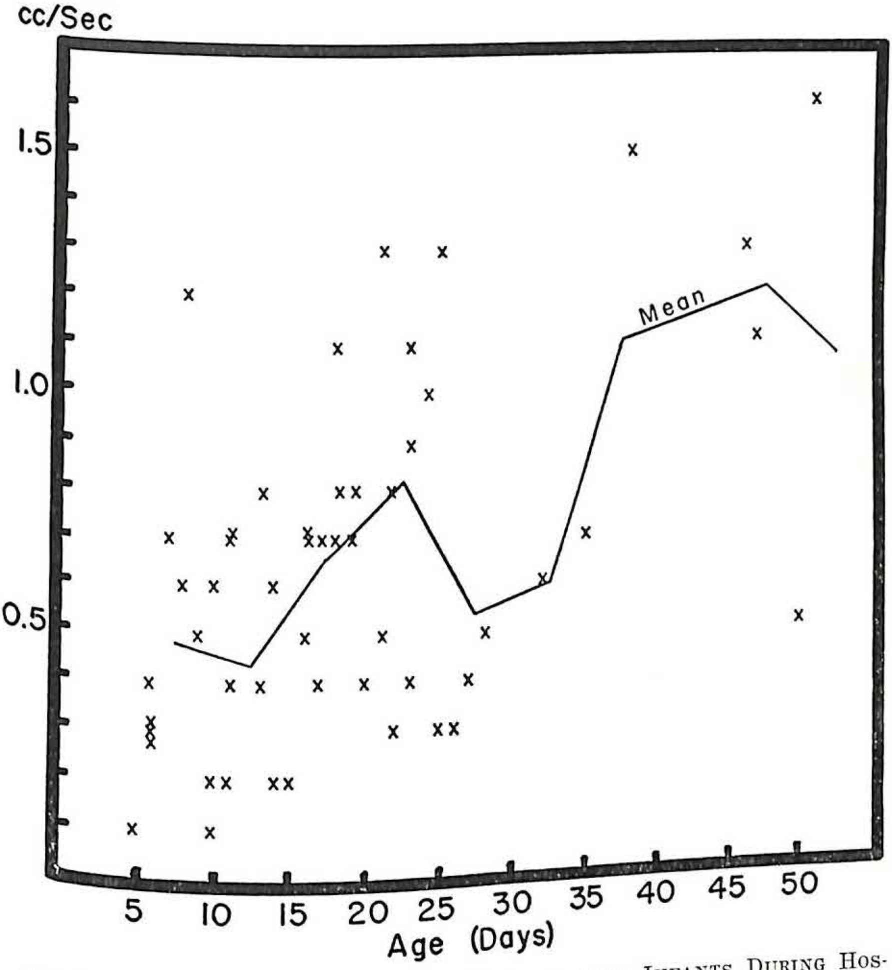

FIG. 7. MAXIMUM SUCKING RATE OF 52 PREMATURE INFANTS DURING HOSPITALIZATION PERIOD, BY POSTNATAL AGE AT WHICH MAXIMUM WAS ACHIEVED.

1. Gestational age and fastest sucking rate during the first three days of formula feeding.
2. Gestational age and the postnatal age at which the maximum sucking rate was achieved.

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[3] Gestational age was secured from clinical record of mother and was generally based on her statement regarding last menstrual period.

The lack of significant association between gestational age and maximal sucking rate during the first three days of formula feeding is somewhat surprising, inasmuch as sucking rate and birth weight were found to be related. It is perhaps possible that the small number of cases studied obscured the relationship. On the other hand, it is well known that gestation period estimates are open to many serious inaccuracies, and that, not infrequently, such estimates do not seem to correspond with the birth weight of the infant nor with the apparent fetal age.

SUMMARY AND CONCLUSIONS

1. The maximal sucking rate of 52 Negro female premature infants and 32 normal newborn Negro infants was studied.
2. It was found that during the first three days of life, premature infants ingested water at a mean maximal rate of 0.16 cc per second. The heavier babies demonstrated a stronger and more effective sucking reflex and thus achieved a higher maximal rate of efficiency.
3. When compared with the normal newborn during the first week of life, the premature infant showed a sucking rate (0.24 cc per second) averaging less than 50% of that of the full-term baby (0.6 cc per second).
4. Unlike the premature, the full-term baby during the first three days of life showed no relationship between his maximal sucking rate and his weight at birth.
5. As postulated, the sucking efficiency of the premature infant appears to follow a regular course of development with advance in postnatal age, the smallest infants generally achieving a maximal rate significantly later than larger babies. Infants whose birth weight was less than 2000 grams required, on the average, 30 days to show maximal efficiency, while those weighing 2000-2500 grams required 15 days.
6. No relationship was found to exist between gestational age and the maximal sucking efficiency of the 23 premature infants on whom complete data are available.

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Correction to Philip Levine's article, "The Influence of the ABO System on Rh Hemolytic Disease," which appeared in the February 1958 issue (Vol. 30, No. 1):

The 17 cases listed in table 1 (p. 16) as "antibodies" are erroneously referred to in the Addendum as "antibodies but no hemolytic disease." The original table in the paper by Owen, *et al.* (*Proc. Nat. Acad. Sci.*, *40*: 420-424), stated that the findings were "based on antibody determinations only," but actually (p. 423) there was severe hemolytic disease in 6, mild disease in 2; 4 infants were apparently not affected, and one mother was still pregnant. As anticipated, the findings in the two contrasting groups totaling 51 cases revealed an excess of ABO compatible matings in the 17 cases (13 ABO compatible, three incompatible, one no data) and an excess of ABO incompatible matings in the 34 cases with no antibodies (18 compatible and 16 incompatible).

DATA FOR QUANTITATIVE GENETICS IN MAN. BIRTH WEIGHT IN OFFICIAL STATISTICS

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INTRODUCTION

THE genetical implications of birth weight as a metrical character have received attention in recent times by Karn and Penrose ('51), McKeown and Gibson ('51), Penrose ('54), Robson ('55), Morton ('55), Fraccaro ('56), Haldane ('57) and others. These studies were based usually on two types of material, either on a series of births in consecutive years from one maternity clinic or on all births in a town in a given time unit. In the first type the information is definitely relevant but the size of the sample is necessarily rather small, e. g., 13,730 in the study of Karn and Penrose ('51); 5,486 in that of Fraccaro ('56). In the second, the size of the sample is larger, e. g., 23,970 births in Birmingham in 1947 as assembled by McKeown and Gibson ('51). A special case is the Japanese data assembled by the Atomic Bomb Casualty Commission (cf. Neel and Schull, '56), which have also been discussed by Morton ('55).

It is possible at this time to extract a very large sample from Czechoslovakian official statistics, which contains information on birth weight of all new born babies, classified by vitality and sex. My attention was called to these data during the U. N. O. World Population Conference in Rome in 1954, when the Czechoslovakian delegation presented a short summary of similar data assembled in 1950 (Proc. World Pop. Conf., '54). I found it of some interest to explore what kind of information could be extracted from this material. I obtained the data in the form presented here through the courtesy of Dr. Frantisek Fajfr.

THE MATERIAL

Information was obtained for a total of 167,731 births which took place in the Czech territory of Czechoslovakia (*i. e.*, former Bohemia, Moravia and Silesia) in 1955. These births include the following categories:

(a) fetuses after 28 weeks of gestation with a minimum length of 35 cm and a minimum weight of 1000 gm,

(b) fetuses not complying with (a) but live born and surviving at least 24 hours,

(c) fetuses complying with neither (a) nor (b) but produced by a multiple birth of which at least one fetus complies with (a) or (b).

Sex distribution and the number of live-births and stillbirths are given for different birth weight groups, divided into class intervals of 500 gm. The distribution of parity (order of birth) and mother's age for different weight groups is also listed, but this material is not divided by sex. Parity and maternal age are not listed concurrently for each birth weight class. The birth order is based on all previous live and still births. Ninety-two per cent of the sample is said to be reported from maternity clinics. The information obtained is given in table 1.

ANALYSIS

Table 2 gives the means and standard deviations for birth weight according to sex and vitality. As expected the stillborn babies are lighter even though those weighing less than 1000 gm have been excluded. This exclusion makes the mean higher than expected. Comparison with other data regarding this point is difficult. For example, the English and Italian data, assembled by Karn and Penrose ('51) and Fraccaro ('56), included both stillbirths and neo-natal death without the weight limitation of 1000 gm under the definition of non-survivors. The mean parity (first-born $= 0$) and mother's age are 1.33 (S. D. $= 1.53$) and 26.94 (S. D. $= 5.57$) respectively. The common anomaly of the prevalence of first-borns is not found in this large sample, in which the mean parity is much higher than in the smaller samples (cf. mean parity 0.90 for all births in Karn and Penrose and 1.05 in Fraccaro). The information obtained has listed maternal age and parity separately as related to birth weight. Had both these variables been listed for each individual birth weight simultaneously, the analysis could have been taken further

TABLE 1

*All births in the Czech territory (former Bohemia, Moravia and Silesia), 1955 **

		BIRTH WEIGHT											
CLASS LIMITS		-999	1000-1499	1500-1999	2000-2499	2500-2999	3000-3499	3500-3999	4000-4499	4500-4999	5000-	UN-KNOWN	TOTAL
Live-births	Males	46	470	1,093	2,975	11,603	31,416	27,054	9,268	1,443	190	22	85,580
	Females	76	472	1,075	3,470	15,427	34,033	20,260	4,813	575	65	28	80,294
Still-births	Males	7	143	158	138	172	189	130	56	10	10	3	1,016
	Females	8	122	138	127	146	162	91	27	13	5	2	841
Parity	0	40	453	980	3,051	12,965	25,467	13,969	2,784	271	32	11	60,023
	1	38	313	671	1,633	7,252	20,446	16,266	4,961	651	81	12	52,324
	2	24	184	399	921	3,543	10,162	8,717	2,946	457	55	7	27,415
	3	20	125	193	509	1,649	4,836	4,210	1,645	265	37	9	13,498
	4	5	60	84	273	887	2,320	2,009	804	167	19	6	6,634
	5	4	29	61	144	439	1,166	1,023	436	96	18		3,416
	6	2	14	37	72	270	632	585	251	49	10	4	1,926
	7	2	13	11	47	145	321	325	127	30	3	1	1,025
	8	1	7	9	23	72	186	189	96	22	4		609
	9 and over	1	9	19	37	125	263	242	114	33	11	2	856
	unknown					1	1					3	5
Maternal age	-15		2	1	3	6	10	3			1		26
	15-19	9	91	272	762	2,715	5,151	2,600	499	51	6	3	12,159
	20-24	41	417	824	2,467	10,741	24,346	16,019	4,004	453	49	16	59,377
	25-29	47	344	642	1,724	7,440	19,741	15,406	4,753	681	74	21	50,873
	30-34	25	204	451	1,087	4,358	11,459	9,370	3,273	550	81	4	30,862
	35-39	8	102	162	416	1,338	3,352	2,788	1,092	198	34	2	9,492
	40-44	7	41	99	235	686	1,620	1,256	496	103	25	5	4,573
	45-49		4	13	15	57	115	90	47	5			346
	50 and over		1		1	4	1	3					10
	unknown		1			3	5					4	13
TOTAL		137	1,207	2,464	6,710	27,348	65,800	47,535	14,164	2,041	270	55	167,731

* The information is in the form obtained.

and the individual contribution of the two variables been estimated. The correlation between birth weight and parity, as calculated from table 1, is 0.112 ± 0.0022, and that between birth weight and mother's age is 0.090 ± 0.0024. The latter is generally reduced when corrected for parity, which is not possible here, since the correlation between age and parity is not available.

TABLE 2

Means and standard deviations of weight for live-births and still-births and their total*

SEX OF INFANT	VITALITY	NO.	BIRTH-WEIGHT (gm) MEAN	S. D.
Male	Live-births	85,558	3,412.09 ± 1.90	556.65
	Stillbirths	1,013	2,649.31 ± 29.89	951.25
	Total	86,571	3,403.17 ± 1.93	568.80
Female	Live-births	80,266	3,274.99 ± 1.84	521.35
	Stillbirths	839	2,571.81 ± 31.71	918.40
	Total	81,105	3,267.71 ± 1.87	531.80
Both sexes	Live-births	165,824	3,345.73 ± 1.34	544.20
	Stillbirths	1,852	2,614.20 ± 21.78	937.30
	Total	167,676	3,337.65 ± 1.36	555.35

* The standard deviations given in tables 2-4 are adjusted with Sheppard's correction.

TABLE 3

Mean birth weight for given parity (all births)

PARITY	NO.	MEAN	S. D.
0	60,012	3,220.50	520.70
1	52,312	3,388.52	538.45
2	27,408	3,409.84	563.15
3	13,489	3,420.40	593.40
4	6,628	3,419.28	599.85
5	3,416	3,427.00	624.80
6	1,922	3,427.68	619.65
7	1,024	3,419.43	632.40
8	609	3,483.99	651.15
9 and over	854	3,435.01	683.50
TOTAL	167,674	3,337.15	554.50

Tables 3 and 4 show the mean birth weight values and their standard deviations for each parity and maternal age. The mean birth weight increases with parity. The increment is more evident between the first and the second birth, when the mean weight increases by 0.3 of σ, and it is still significant between the second and the third births. In the higher parities the mean weights remain rather constant.

TABLE 4

Mean birth weight for given maternal age (all births)

MATERNAL AGE	NO.	MEAN	S. D.
-15	26	2,942.31	812.30
15-19	12,156	3,180.53	532.65
20-24	59,361	3,296.85	531.80
25-29	50,852	3,376.11	547.85
30-34	30,858	3,396.87	570.20
35-39	9,490	3,386.88	613.15
40-44	4,568	3,360.00	632.65
45-49	346	3,332.37	648.90
50 and over	10	2,900.00	
TOTAL	167,667	3,337.67	555.35

According to previous studies (among others, Millis and Seng, '54; Neel and Schull, '56) the relationship between birth weight and parity is consistent and generally of a curvilinear type, while mother's age has a negative effect when parity effects are removed. The parabolic distribution of mean birth weights for given mother's age which is observed in table 4 is probably not real. Unfortunately the official statistics do not provide weights for given mother's age and parity, and therefore they do not allow an efficient analysis on this point. It is worth noting, however, that while Millis and Seng ('54) observed in their material that mean birth weights at different maternal ages for all parities are fitted by a straight regression line (cf., figure 4, p. 67 in their paper), in the present data the mean birth weights at different maternal ages as given in table 4 are fitted by a parabola.

Table 5 shows the stillbirth rates for various birth weight groups for males and females respectively. The stillbirth rate is higher in males, the females being lower by 0.14 ± 0.05%. The overall stillbirth rate in the sample is 1.10%. The fact that stillborn babies weighing less then 1000 gm are not included should be remembered in judging these rates. The heavy elimination of the very light and very heavy babies by stillbirth is evident from table 5. The babies weighing between 3000 and 4000 gm have the best chance of being alive at birth, while only about 80% of the babies in the weight group 1000-1500 gm are live births. An estimate of the birth weight associated with optimal vitality at birth is given by the ratio

TABLE 5

Still birth rates for given birth weight

BIRTH WEIGHT (gm)	MALES			FEMALES		
	ALL BIRTHS	STILL BIRTHS	STILL BIRTH RATE (per cent)	ALL BIRTHS	STILL BIRTHS	STILL BIRTH RATE (per cent)
- 999	53	7	13.21 ± 4.65	84	8	9.52 ± 3.20
1000-1499	613	143	23.33 ± 1.71	594	122	20.54 ± 1.66
1500-1999	1251	158	12.63 ± 0.94	1213	138	11.38 ± 0.91
2000-2499	3113	138	4.43 ± 0.37	3597	127	3.53 ± 0.31
2500-2999	11775	172	1.46 ± 0.11	15573	146	0.94 ± 0.08
3000-3499	31605	189	0.60 ± 0.04	34195	162	0.47 ± 0.04
3500-3999	27184	130	0.48 ± 0.04	20351	91	0.45 ± 0.05
4000-4499	9324	56	0.60 ± 0.08	4840	27	0.56 ± 0.11
4500-4999	1453	10	0.69 ± 0.22	588	13	2.21 ± 0.61
5000-	200	10	5.00 ± 1.54	70	5	7.14 ± 3.08
TOTAL	86571	1013	1.17 ± 0.04	81105	839	1.03 ± 0.04

$$\frac{m_{LB} \times V_{SB} - m_{SB} \times V_{LB}}{V_{SB} - V_{LB}},$$

where m and V are the mean and the variance of livebirths (LB) and stillbirths (SB) (cf. Fraccaro, '56).

These estimates are 3809 gm for males and 3609 gm for females. They are higher than the means by 0.7 and 0.6 of a standard deviation unit, respectively. The same estimates calculated using the variance unjusted for grouping effect are 3836 gm and 3635 gm respectively. I have discussed the selective importance of differential mortality associated with birth weight in a previous paper (Fraccaro, '56), and further discussion on this point can be found in Haldane ('57) and McKeown ('57).

CONCLUSION AND SUMMARY

The data on birth weight of 167,731 births in the Czech territory included in the Czechoslovakian official statistics have been briefly examined. Mean birth weight is higher in males than in females for both categories of live- and stillbirths. As expected, stillborn babies are lighter than liveborn.

Stillbirth rates for given birth weight groups have been calculated. They show the selective effect of birth weight at the extremes, where very light and very heavy babies show high stillbirth rates. The overall stillbirth rate is 1%. The stillbirth rate for the birth weight group 1000-1500 gm is about 20% and for the weight group above 5000 gm about 6%.

An estimate of the birth weight associated with the optimal vitality at birth, gives values above the mean.

The effect of parity of the mother and maternal age on birth weight cannot be efficiently estimated from the present data, which unfortunately do not give the two variables for any birth weight group simultaneously. However, mean birth weights for given parity and mother's age have been calculated and presented.

The introduction into official statistics of more biological data of the nature reviewed in this paper is to be warmly welcomed, although such data are not always fully suitable for immediate fruitful analysis. Their accumulation will, however, prove of extreme value to future students.

ACKNOWLEDGMENT

I wish to acknowledge the courtesy of Dr. Frantisek Fajfr, president of the National Statistical Office, Praha, who kindly provided the data discussed in the paper. Thanks are also due to Mr. E. Lander, Uppsala, for help with some of the calculations and to Dr. K. Hirschhorn, New York, for reading the manuscript.

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REFLECTOMETRY OF THE SKIN IN SOUTHERN NIGERIANS AND IN SOME MULATTOES *

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INTRODUCTION

REFLECTOMETRIC techniques offer a more objective method for the quantitative study of pigmentation than do the earlier visual scoring and matching procedures. Edwards and Duntley ('39a) and Sheard *et al.* ('26) explored the reflectometry of normal human skin, and the method has also found a use in work on sunburn (Edwards and Duntley, '39b, Jansen, '53) and in endocrinological studies (Lerner *et al.*, '54, Edwards *et al.*, '41; Edwards and Duntley, '49).

The recent introduction of inexpensive, portable reflectometers has opened up the possibility of population surveys for anthropological purposes and of more accurate work on the genetics of pigmentary differences. Lasker ('54) has reported some data on a Mexican population, and pigmentary changes with age have been studied by Garn *et al.* ('56) who examined several different skin areas in white subjects of both sexes. Harrison ('57) has given an account of his work on Negro-European hybrids.

The present paper, based on material collected in Southern Nigeria in January-April, 1956, deals with the skin color of two indigenous peoples who are compared with one another and with Europeans, and also with the skin pigmentation of a small sample of African-European hybrids, mainly from Warri in the Niger Delta region.

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* The tables of original data for this investigation have been deposited as Document number 5532 with the ADI Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D. C. A copy may be secured by citing the Document number and by remitting $1.25 for photoprints, or $1.25 for 35 mm microfilm. Advance payment is required. Make checks or money orders payable to: Chief, Photoduplication Service, Library of Congress.

THE SAMPLE

One hundred Yoruba males and 94 Yoruba females attending the general medical clinic at the Adeoyo Hospital, Ibadan, were measured. Their ages, which were only known approximately, ranged from 18 to 60 years. The majority (101) of the Yoruba subjects gave Ibadan as their birthplace; 30 were born in Ijebu; 17 in Abeokuta; and the remainder in various other parts of Yoruba country. Fifty male Ibo students at the University College, Ibadan were also measured; 21 of these came from Onitsha; 11 from Owerri; and 8 from Awka. Finally, measurements were also made on 14 male Yoruba students from the University, Ibadan.

The European sample which was measured in London a year later, and therefore in the winter months, comprised mainly academic staff, students and technicians at University College, London. Two persons of German birth, one Italian, and one Swede were also included.

TECHNIQUE

A portable Evans Electroselenium Company reflectometer (Weiner, '51) was used and the measurements were taken on the flexor surface of the right forearm at the junction of the middle and proximal thirds. Tatoo marks had to be avoided in the Yoruba women. The site was cleaned by gently wiping with cotton-wool soaked in soapy water. A magnesium carbonate block, which could be periodically cleaned, served as a white standard and readings were taken with 9 color filters, the transmission maxima of which are shown in table 1. To check the performance of the instrument readings on gray paper standards at filters 601 (blue) and 609 (red) were interspersed with the skin readings. During the later part of the work, a second, somewhat lighter standard was established and was also used in England. Successive readings on these standards showed no significant trend over the period of the experiments, and agreement between readings in Nigeria and in England was close. The standard deviations of the sets of readings obtained on the two standards in both countries varied between 0.52% and 0.66% and were closely similar at the two ends of the spectral range. The instrumental error is therefore small in relation to readings on European skin, but may amount to some 8% of the reading on African skin in the blue end of the spectrum. It must be remembered that the quite small errors arising from variations in pressure when placing the photometer-head on the arm are not included in the readings on the standards.

The work at Ibadan was done in air-conditioned rooms and the instrument behaved satisfactorily, but in the high temperatures and humidities of the Delta, deterioration of the photocell occurred if it was exposed for too long to the air. This trouble was met by keeping the photocell-head in a dessicator when not in use and minimizing the exposure to humid atmospheres.

It is perhaps worth emphasizing that the reflectance curves obtained with different types of instrument will not necessarily be the same since differences in the optical system, in the characteristics of the filters, if such are used, and in the light source may cause the results to diverge. Comparing, for example, the curves for the skin of the same European subject on the Evans Electroselenium Company instrument and the General Electric Company Hardy recording spectrophotometer, we found the readings with the former to be about 25% higher at 435 mμ and about 4% lower at 685 mμ.

RESULTS

In table 1 the mean reflectances at each wave length and the standard deviations are set out for each of the various samples, and the results are shown graphically in figure 1. The mean values of reflectance at each wave length constitute the basic data from which indices of overall lightness and darkness and hue can be derived; they are also the essential data for the calculation of energy absorption in the visible range. The mean of the readings at 9 wave lengths may be taken as an approximate measure of overall lightness/darkness irrespective of the particular pigments or optical effects concerned, and the values of these means are also given in the tables.

As a measurement of pigmentation due to melanin the reading at 685 mμ (filter 609) is the most suitable, since as Jansen ('53) and others have pointed out, the amount of oxygenated blood in the skin affects the readings very little in the red region. We found that venous congestion with a sphygmomanometer cuff may lower the reading at this wave length by about 10% in a European; however, these are extreme conditions and in ordinary circumstances variations in blood supply are likely to have much less effect; in Africans, due to masking of the blood by melanin, they are even less important.

TABLE 1.

Mean percentage reflectance and standard deviation (in parentheses) at 9 filters for African and European samples

SEX	SAMPLE	NO.	WAVE LENGTH OF TRANSMISSION MAXIMUM AND FILTER NO.									MEAN REFLECTANCE NINE FILTERS
			601 426 mμ	602 470 mμ	603 490 mμ	604 520 mμ	605 550 mμ	606 580 mμ	607 600 mμ	608 660 mμ	609 685 mμ	
MALE	Yoruba Med. Clin.	100	8.0 (0.91)	8.3 (1.00)	8.1 (1.17)	9.7 (1.43)	10.1 (1.15)	10.9 (1.54)	14.3 (3.08)	20.6 (3.51)	23.6 (3.79)	12.6 (1.84)
	Yoruba Students	14	7.9 (0.67)	7.7 (0.56)	7.7 (0.58)	9.1 (0.59)	9.3 (0.78)	10.1 (0.85)	13.2 (1.22)	19.6 (1.91)	22.9 (2.05)	11.9 (0.93)
	Ibo Students	52	8.7 (1.12)	9.2 (1.23)	9.3 (1.40)	10.7 (1.56)	11.2 (1.73)	12.6 (2.15)	17.0 (3.14)	24.6 (4.05)	28.2 (4.14)	14.6 (2.19)
	Europeans	50	32.8 (3.84)	39.7 (3.99)	41.4 (4.07)	41.6 (3.90)	37.9 (3.72)	41.4 (3.64)	52.4 (4.05)	59.9 (2.90)	61.5 (2.77)	45.4 (3.37)
FEMALE	Yoruba Med. Clin.	94	8.5 (0.80)	8.9 (1.07)	8.9 (1.19)	10.5 (1.33)	11.1 (1.83)	12.2 (2.02)	16.2 (2.85)	23.1 (3.64)	26.1 (4.17)	13.9 (2.04)
	Europeans	50	34.3 (3.48)	41.4 (3.93)	43.1 (3.90)	43.9 (3.76)	40.5 (3.79)	43.9 (3.55)	54.3 (2.95)	61.5 (2.72)	63.1 (2.34)	47.3 (3.09)

The distributions of the readings at 685 mμ are shown in figure 2. The African distributions are somewhat skewed, which is to be expected at the lower end of the percentage scale, but it was not considered necessary to correct for this in making statistical comparisons. The relation between the reflectance and the actual amount of melanin in the skin would have to be established empirically since the optical properties of skin are complex and the application of simple absorption laws may not be justifiable.

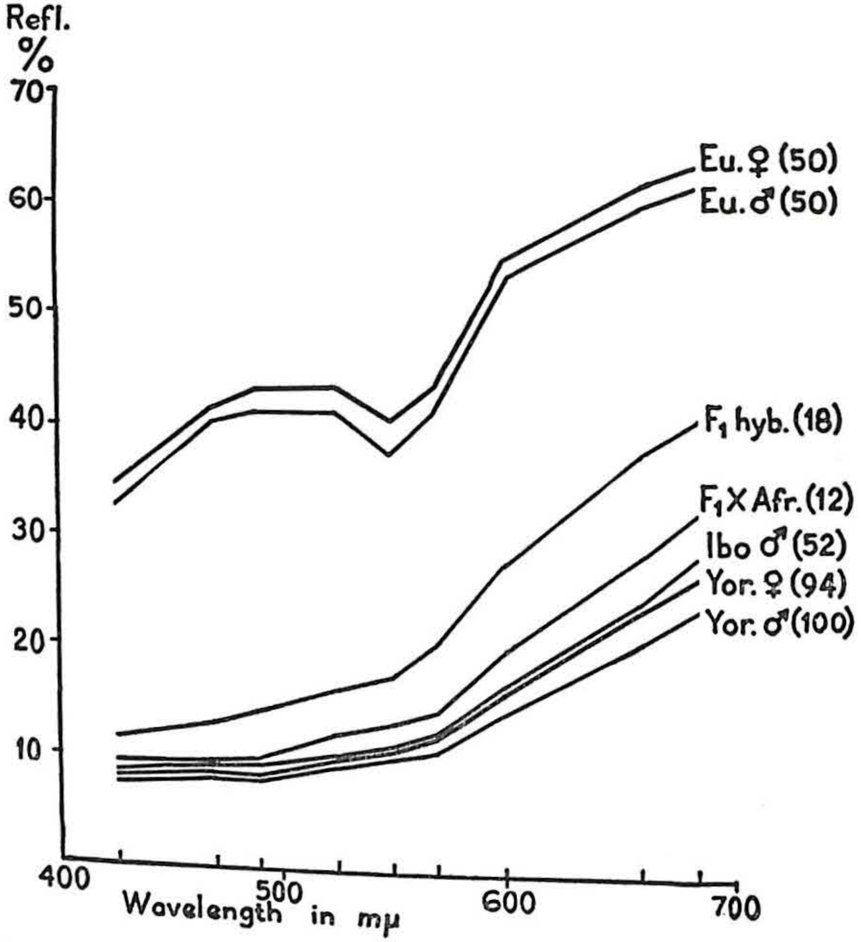

FIG. 1. CURVES DRAWN THROUGH THE MEAN REFLECTANCE VALUES AT EACH OF 9 WAVE LENGTHS FOR EUROPEAN, AFRICAN AND HYBRID SAMPLES.

The curves for European males and females are similar in shape but the reflectance of the males is lower at all wave lengths. The males

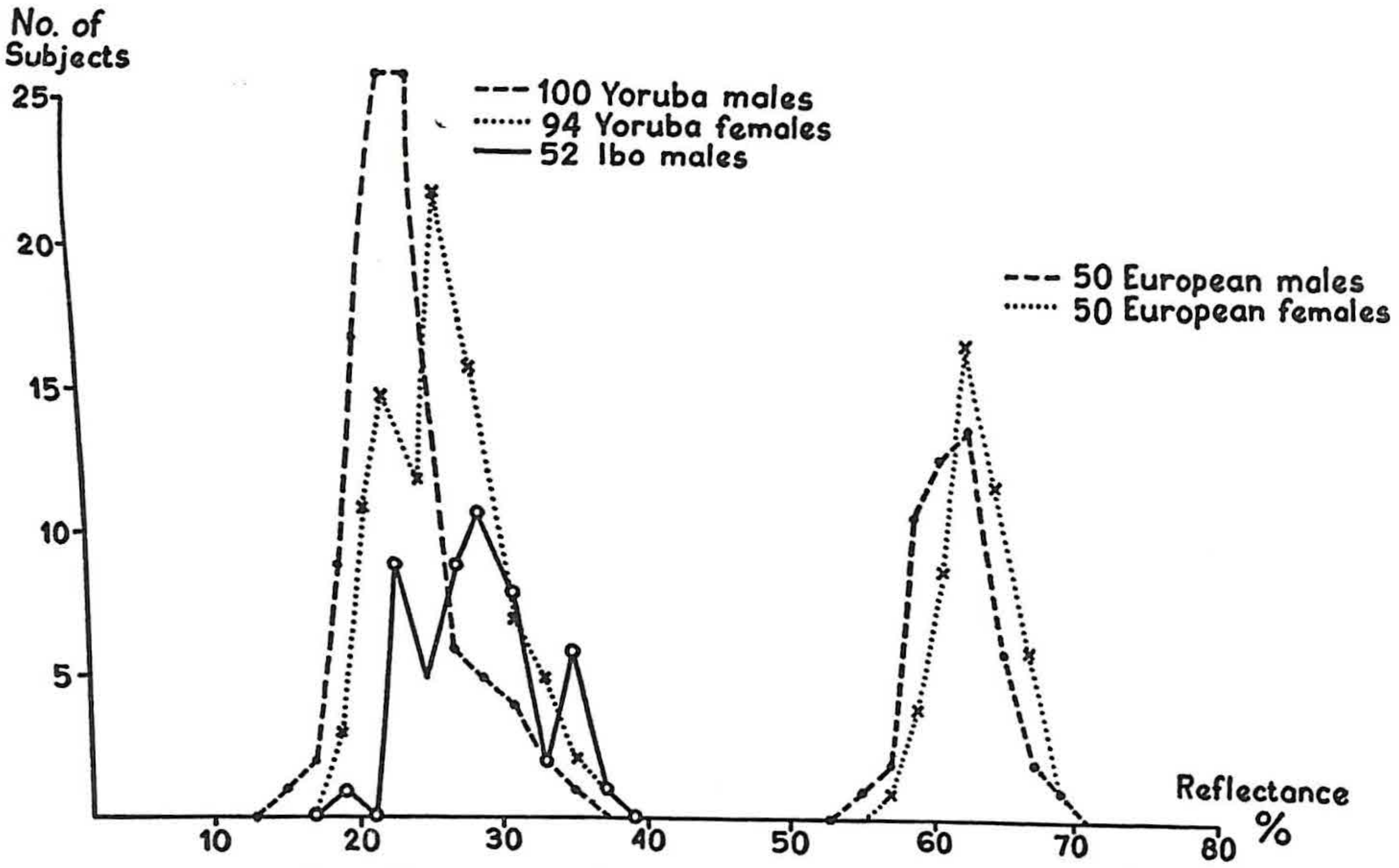

FIG. 2. DISTRIBUTIONS OF PERCENTAGE REFLECTANCE AT 685 mμ (Filter 609) FOR AFRICAN AND EUROPEAN SAMPLES.

also show somewhat greater variability at all wave lengths. It will be noticed that in both sexes the variability, compared as the coefficient of variation, is very considerably greater in the blue and green regions than in the red, due presumably to the fact that variations in the blood supply exert their main effects in this region. Comparing the male and female readings at 685 mμ we find "t" for 98 d. f. is 2.32, significant at the 5.0% level. The more superficial position of the large veins and the presence of dark hair on the arm in some male subjects must be taken into account in interpreting this sex difference in reflectance. Garn *et al.* ('56) also noted a tendency for the reflectance of male subjects to be lower; their measurements, however, were made at 420 mμ only, a region in which not only melanin but also haemoglobin absorbs strongly, and the effects of the two pigments are therefore seriously confused.

Lower reflectances in the males were also found in the case of the Yoruba samples; again comparing the mean readings of the two sexes at 685 mμ, we obtain "t" for 192 d. f. equals 3.09 which is significant at the 1.0% level. This difference may be due at least in part to differences in sun-tan, since this part of the arm is more often exposed in African males than in females.

Comparing the Yoruba males from the medical clinic with the Ibo students in the same way, we obtain "t" $= 4.76$ for d. f. 148, which is significant at the 0.1% level, thus confirming local opinion that the Ibos are the lighter skinned. The variance of the Ibos is greater but not significantly so. The Ibo students may also be compared with the Yoruba students. Due to the rather lower mean and considerably lower variance of the latter as compared with the medical clinic sample, we again obtain a significant difference ("t" $= 3.77$, d. f. 62, $P = 0.001$).

Hybrid samples

The measurements on hybrid subjects were mostly obtained in the towns of Warri and Sapele. The results are summarized in table 2. They comprise 18 adults who were the children of matings between an African mother of the Jekri tribe (except in one case) and a European father who was generally English or Scottish. In some cases these hybrids, who may be regarded as an F_1, had children by an African or by another F_1 hybrid, and the results for a few offspring of such crosses are also shown in table 2.

The Jekri people have a long history of miscegenation with Europeans with whom they came in contact as traders. Local feeling places no obstacle in the way of obtaining details of the pedigree in these cases. We were particularly fortunate in having the assistance of Miss O. A. Igrakpata, the Senior Social worker at Warri, the town in which she was born, in obtaining the full cooperation of the families. According to notes on the social position of mulattoes kindly supplied by Mr. P. Lloyd of the Institute of Social and Economic Research, Ibadan, the majority of the mulattoes are offspring of their mother's first liaison and are often the eldest and only children, with whom the mother comes to live in her old age. Some marry Africans and are fully accepted into African society, and most of them are prosperous and become wealthy by trading or by running sewing-schools, etc.

Unfortunately, because of the effects of the climate on the instrument and shortage of time, it was impossible to measure an adequate sample of Jekri women to represent the maternal side of these crosses. There is no reason to think, however, from what little work we were able to do and from general observation, that the Jekri differ in pigmentation from the Yoruba who are linguistically related to them.

The mean reading at 685 mμ for the F_1 hybrids is 41.0 (table 2), which is close to the mid-value (43.8) between the means of the Yoruba females and European males, who may be taken to represent the parental populations. It must be remembered, however, that the mean of the latter would doubtless be lower if they had been exposed to sunshine to the same extent as the Nigerians. The variance of the F_1 hybrids (28.27) is substantially higher than that of the Yoruba females (17.38), but the coefficients of variation are similar. The mean of the "backcross" of F_1 hybrids to Africans (table 2) is 32.3, which is close to the mid-value of 32.0 between F_1 hybrids and Yoruba males, while the mean of F_2 offspring (table 2) is 39.6 which is almost the same as the F_1 mean (41.0), but there is no evidence of increased variance.

The data are insufficient to justify elaborate biometric analysis and an attempt to find a consistent scale compatible with an additive form of multifactorial inheritance. The percentage reflectance scale necessarily produces bunching of the values at the two ends. It is worth noting that if the raw reflectances are transformed by the expression Log R-log (100-R) as used by Sunderland ('55) in work on hair color, the variance of the Yoruba females (0.0079) is brought close to that of the F_1 hybrids (0.0094) without affecting the relationship between the means of the three samples very much. The variance of the European males (0.0026) remains much lower; the low variance of this sample is no doubt partly due to the minimal exposure to sun-tanning.

TABLE 2

Mean percentage reflectance and standard deviation (in parentheses) of African hybrids

MATING TYPE OF PARENTS	NO. OF OFFSPRING MEASURED		APPROX. AGE RANGE IN YEARS	REFLECTANCES AT 9 WAVE LENGTHS									MEAN OF 9 WAVE LENGTHS
	MALE	FEMALE		426 mμ	470 mμ	490 mμ	520 mμ	550 mμ	580 mμ	600 mμ	660 mμ	685 mμ	
European male X African female [1]	4	14	30-65	12.0 (2.13)	13.1 (2.45)	14.2 (2.99)	16.4 (3.34)	17.6 (3.76)	20.3 (4.37)	27.9 (5.48)	37.4 (5.64)	41.0 (5.36)	22.4 (3.79)
F_1 hybrid X African [2] (5 families)	4	8	6-25	9.5 (1.30)	9.8 (1.51)	10.0 (1.94)	12.4 (2.40)	13.1 (2.60)	14.4 (3.42)	20.0 (4.81)	28.1 (6.66)	32.3 (6.00)	16.5 (3.36)
F_1 hybrid X F_1 hybrid (4 families)	2	5	One 48 remainder 3-10 yr.	12.0 (1.67)	12.4 (2.00)	13.6 (2.04)	15.7 (2.38)	17.1 (2.91)	20.6 (3.65)	27.4 (4.28)	35.8 (5.06)	39.6 (4.94)	21.4 (3.19)

[1] All the African mothers were Jekri except one who was Ibo. The European fathers were English (7), Scottish (7), German (2), Portuguese (1), Lebanese (1).

[2] In all cases except one, one parent was a Jekri-European hybrid and the other Jekri; in one case the father was Jekri-German and the mother Urhobo.

The assumptions involved in interpreting results on material of this kind should be carefully examined. It is not improbable that Africans and Europeans have some pigmentation genes in common so that evidence of segregation might be apparent in the variance of the first generation hybrids and would decrease the difference of variance between this generation and the F_2, which is a classical criterion of mendelian effects in experimental material. The influence of environmental factors, in particular sun-tanning, on the mean and variance may well be different between hybrids and the general population, since F_1 hybrids tend to enjoy a more sedentary indoor existence. It seemed likely that one or two of the male subjects were sun burned, however, so that the range of variation may be unusually great. One cannot safely assume, as Kuppenheim and Heer's ('52) work seems to indicate, that the relative effect of sun-tanning will be the same at all levels of initial pigmentation. The likelihood of some degree of assortative mating should be borne in mind when dealing with a character such as skin color, but the close correspondence between the means of the hybrids and the mid-parental values suggests that this factor has not been important in the present case.

SUMMARY

1. The reflectance of the skin on the flexor surface of the forearm in some African and European subjects has been measured at nine filters covering the visible spectrum.
2. The reflectance is found to be lower in male subjects, both African and European.
3. The Yoruba of Southwestern Nigeria are shown to be somewhat darker than Ibo from the Southeast.
4. Some reflectance data on the offspring of crosses between Europeans and Africans (F_1) and of certain other crosses is presented as a contribution to the genetics of skin color differences.

ACKNOWLEDGMENTS

The work was done during a visit to Nigeria financed by a grant from The Rockefeller Foundation. I am grateful to the Department of Medicine, University College, Ibadan, for providing laboratory accommodation and also some of the subjects, and to Professor C. W. L. Bevan, Professor J. Grayson and Mr. R. O. Thomas for providing access to others. I am also indebted to Mr. Preston of the National Physical

Laboratory for advice regarding reflectometry and to my wife for assistance in the field investigations.

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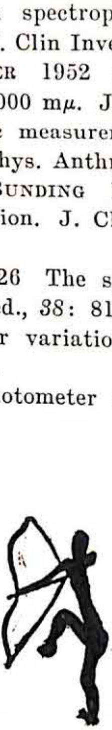

BOOK REVIEWS

Sports in the Cultural Pattern of the World: A Study of the 1952 Olympic Games at Helsinki. By Ernst Jokl, M. J. Karvonen, Jaakko Kihlberg, Aarni Koskela and Leo Noro. VII + 116 pp. Helsinki: Institute of Occupational Health, 1956. No price indicated.

The chronic effects of physical exercise are of very real interest to students of growth and development and of other aspects of human biology. There is an urgent need for longitudinal investigations on such fundamental problems as the consequences, if any, of athletic participation of girls on the development of the pelvis, asserted to be narrower in female athletes. Only longitudinal studies will help to separate the factors of selection from the real somatologic effects of athletics.

In the present volume a group of scientists examined Olympic participation and athletic performance in reference to the characteristics of the individual athletes (age, sex) and of the countries (such as climate, death and birth rates, and nutrition) which they represented at the Summer Games.

Methodologically, of interest is the novel method of allocating points for various Olympic performances, based on ranking the competitors. The winner is assigned 100 points, the last man 0 point, with the intermediate point scores (P) assigned on the basis of the formula $P = 100 (1 - \log x/\log n)$, where x = ordinal number of the competitor, n = the number of competitors in an event. When n is given, P is a linear function of the logarithm of the competitor's rank (x). Examples of the point allocation system are given both in tabular and graphic form for groups varying in the number of participants.

A wealth of data is presented in the text and in the appendixes, including the number of participants from the various countries, actual and related to population size (participation rate); number of points collected, given as absolute figure (point share), as points per million inhabitants (point rate), and as average number of points collected in the events in which a country's representatives participated (point level).

Olympic participation and achievement is presented in reference to eight geographical regions, with the highest point level attained by the U. S. S. R. (41.6) and United States plus Canada (37.1). The highest point rate was held by Europe (124.5); the corresponding value for North America was 64.6, for the U. S. S. R., 45.0.

As to sex, the high standards of physical performance of women, attested at Helsinki, leads the authors to call for a re-examination of the traditional reserve with which the physical training of women is frequently treated by educators and physicians. The authors believe that scientific studies of women's athletics indicate that biologically the female sex is more robust and more capable of adjusting to the demands of physical activities than has been generally assumed. The extension of the age span of women athletes participating in top-level competition, noted at Helsinki, is interpreted as reflecting the process of acceleration of growth and of deceleration of aging.

Paragraph 40 of the Olympic Rules specifies that "There is no age limit for competitors in the Olympic Games." The age range of the participants—from boys and girls of 13 to 14 to women of 45 and men of 66 years—is truly amazing. Different competitive events show distinct age patterns; thus male swimmers averaged 21.5 years while the median age of competitors in shooting events was 37 years. The authors are careful to point out that average ages of top-level performers can in no way serve as indicators of the suitability of the particular sport for purposes of attaining or maintaining physical fitness.

In regard to relation between racial background and performance, the White race was dominant in the longer runs and in the throwing events while the Negro race played a major role in the shorter runs. The representatives of Asiatic populations, though few in number, were successful in many sports events. It is of interest, from several points of view, that the three most outstanding long-distance swimmers—while competing on behalf of the United States, Japan and Brazil—were all of Asiatic racial stock.

Here, and in relating Olympic performance to size of the countries, their climate, vital statistics and economic conditions we deal with complex, partly interrelated factors. The association with performance scores must be interpreted with caution. What is the real significance of the empirically established tendency to a positive correlation between the average caloric consumption per person for a country and the point level attained at the Olympic games, an observation that is considered as "one of the most important results of this survey" (p. 81)?

The volume, though slim, contains a large amount of information, carefully processed statistically, that is of interest to specialists in the scientific study of athletics as well as from a more general biological point of view. There are photographs, 24 tables, 27 figures and 5 appendixes.

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The Aleut Dentition: A Correlative Study of Dental Characteristics in Eskimoid People. By Coenraad F. A. Moorrees. x + 196 pp. Harvard University Press, 1957. $4.50.

The book contains a readable discussion of the various characteristics and problems attendant to dentitions generally. This, together with simple and comprehensive definitions of terms used in comparative odontometry, will appeal to human biologists.

Moorrees' description deals with dental materials obtained by Harvard's Peabody Museum expedition to the Aleutian Islands in 1948. Laughlin, who headed the expedition, considers the contemporary Aleuts as belonging to two breeding isolates, eastern and western. According to Moorrees, Laughlin's hypothesis is based on anthropometric, serologic, ethnologic and linguistic differences between the two groups. The distribution of blood groups confirms the racial origin of the Aleut as Eskimoid, with only a slight degree of white mixture.

Moorrees observes that the dentition offers a distinct advantage for differentiating between eastern and western Aleuts because of the large number of independent characteristics that can be studied. He believes that while the east-west differences reported in his book in no way settle the issue, they provide important material for the evaluation of Laughlin's hypothesis. The *torus mandibularis* is highly prevalent among the Aleuts. Twenty-six percent of the western Aleuts and 61% of the eastern Aleuts had this exostosis, and the regional difference was statistically significant. In both regions, males not only have a greater prevalence of tori, but also a greater frequency of the most pronounced ones. One interesting finding was that no incisors or premolars were found missing in either jaw in the available cases. An absence of one or more third molars, however, occurred in a much larger percentage than is commonly found in other population groups.

In addition to details of morphology, Moorrees discusses problems of nutrition, health and dental caries. With minor variations, the main source of food still is the sea but various birds are also available. Sea-gull eggs, a variety of berries and greens such as wild celery, and the bulbs of a Kamchatka lily, similar in use and composition to the potato, supplement the diet.

The results of the comparison of the average daily food intake for a limited group of Aleut women showed that the protein, fat, vitamin A, niacin and vitamin D intake levels exceeded the NRC standards, due mainly to the use of salmon and seal oil. Moorrees proposes " that the increase in the incidence of dental disease among contemporary Aleuts is associated with an unfavorable change in the quality of their tooth structure. This change in turn may be one of the inevitable by-products of a decline in general health and of rather marked changes in the dietary during the first half of the twentieth century. The great increase in the intake of refined carbohydrates may have placed an additional burden on the already weakened dental tissues by producing certain unfavorable changes in the immediate environment of the teeth. Substantiation of these speculations would require access to factual data that is not available at present."

As the preceding quotations show, Moorrees is extremely conscientious in not giving overemphasis or oversignificance to any of the features which he discusses in his book. In a section at the front of the book, preceding the text, is an interesting assemblage of 41 figures, including charts, photographs, drawings and tables. Scattered through the text are 57 tables which embellish and give substance to the discussion. It is regrettable that the sample size of Aleuts is so small.

This book is well organized and well executed. It was used as one of the texts in the course on Hominid Dentition at the University of Chicago this year.

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BOOK NOTICES

Human Histology. By Leslie Brainerd Arey. ix + 337 pp. W. B. Saunders, Philadelphia, 1957. $6.50. Standard in scope, unorthodox in treatment (a "textbook in outline form") and designedly without illustrations.

Scientific Study of Social Behaviour. By Michael Argyle. xiii + 239 pp. Philosophical Library, New York, 1957. $6.00. Part I deals with methodology, Part II with generalizations and theories of social interaction. The final, large chapter is concerned with human relations in industrial, military and similar social organizations.

Modern Researcher. By Jacques Barzun and Henry F. Graff. xiii + 386 pp. Harcourt, Brace & Co., New York, 1957. $4.50. An unconventional attempt by two historians to combine initiation into techniques of (humanistic) research with the cultivation of the art of expression. It is refreshing to read that facts *never* "speak for themselves"—they must be given an intelligible voice.

Physical and Chemical Basis of Inheritance. By G. W. Beadle. 47 pp. University of Oregon Press, Eugene, Ore., 1957. $1.00. Presented in the Condon Lectures series designed to interpret the results of significant scientific researches to the nonspecialist.

Functional Neuro-Anatomy. By A. R. Buchanan. 362 pp. Lea & Febiger, Philadelphia, 1957. $7.50. Third, revised edition of a textbook presenting economically the essential material. Appended is an atlas of brain stem and adjacent structures (pp. 262-335).

Physical Methods in Physiology. By W. T. Catton. xi + 375 pp. Philosophical Library, New York, 1957. $10.00. Presents, in an abbreviated form, selected techniques applied to physiological investigations of blood, muscle, peripheral nerves and of the processes of circulation, respiration and heat exchange. The concluding chapter discusses electronics in physiology.

Fats in Human Nutrition: Symposium. 131 pp. Council on Foods and Nutrition, American Medical Association, Chicago, Ill., 1957. No price indicated. In recent years, interest in the subject of the symposium (6 papers and panel discussion) has been stimulated markedly by findings suggesting that dietary fat may be implicated in the genesis of atherosclerosis and coronary heart disease, previously considered as an inevitable consequence of aging.

Guide Book to Research Data for Arid Zone Development. Edited by B. T. Dickson. 191 pp. UNESCO, Paris, 1957. $6.00. Concerned with aspects that should be covered in a preliminary survey of arid or semi-arid lands as a basis for development plans. Part I is devoted to physical and biological factors, Part II to human factors. The usefulness of several of the bodily characteristics recommended for study (p. 144) appears to be highly questionable.

BRAINPOWER QUEST. Edited by Andrew A. Freeman. xiv + 242 pp. Macmillan, New York, 1957. $4.75. Proceedings of a symposium on providing adequate engineering and scientific manpower, sponsored by the Cooper Union for the Advancement of Science and Art and attended by educators, scientists and men of affairs.

MOVEMENT OF THE HEART AND BLOOD IN ANIMALS. By William Harvey. xii + 209 pp. C. C. Thomas, Springfield, Ill., 1957. $3.50. The classical Latin work on blood propulsion translated afresh and competently by K. J. Franklin.

EXPERIMENTAL PSYCHOPATHOLOGY. Edited by Paul H. Hoch and Joseph Zubin. x + 275 pp. Grune & Stratton, New York, 1957. $6.50. The proceedings of the 45th Annual Meeting of the American Psychopathological Association represent well the main currents in this field. The collaboration of clinicians and experimentalists, fostered by the Association, is important for bringing about the emergence of a common vocabulary, an experimentally oriented science of abnormal behavior, and a scientifically grounded therapy.

PRE-EMPLOYMENT DISABILITY EVALUATION. By William A. Kellogg. xv + 155 pp. C. C. Thomas, Springfield, Ill., 1957. $10.50. A detailed reference manual for medical examiners. Gives (on p. 86) tables for height and weight, without clothing, for men and women 15, 20, 25 and 30 years old. The means are "adjusted" for three types of body build, without specifying as to how "body build" is determined in actual practice.

BIBLIOGRAPHIE DER SPORTMEDIZIN UND IHRER GRENZGEBIETE 1955 (Bibliography of Sport Medicine and of Neighboring Fields). By E. J. Klaus. 96 pp. Georg Thieme, Stuttgart, 1956. $0.85. References to German publications for the year 1955 with a detailed index.

NOMINA ANATOMICA. By Fr. Kopsch. Revised by K. H. Knese. xi + 155 pp. Georg Thieme, Stuttgart, 1957. $1.55. With the Basel (1895) anatomical nomenclature arranged in alphabetical order as a basis, the terminology accepted at Jena (1935) and revised in Paris (1955) is given in parallel columns.

LIGHT, COLOUR AND VISION. By Yves Le Grand. xiii + 512 pp. John Wiley, New York, 1957. $11.00. Translated from French, this is a detailed account of the eye as a receptor of radiant energy. Section A gives experimental facts, presented from the point of view of the physicist and addressed to those who can apply them (opticians, illuminating engineers, architects). Section B deals with theories of vision.

HISTORICAL BACKGROUND OF CHEMISTRY. By Henry M. Leicester. viii + 260 pp. John Wiley, New York, 1956. $6.00. The development of chemistry is followed in reference to the ideas of chemists rather than their biography. Final chapter is devoted to biochemistry.

SOME APPLICATIONS OF BEHAVIOURAL RESEARCH. Edited by Rensis Likert and Samuel P. Hayes, Jr. 333 pp. UNESCO, Paris, 1957. $3.25. Dr. Likert opens the volume with the manifesto on "Behavioral Research: A Guide to Effective Action." The applications are described with reference to problems of industrial management, training of leaders, marketing and business forecasting.

THE MERCK MANUAL. Edited by Charles E. Lyght. xv + 1870 pp. Merck, Rahway, N. J., 1956. $6.75. This is a physician's manual. Non-medical scientists may find of interest the sections on diets (which includes the perennial "ideal weight" weight tables for individuals of different frame, again without any numerical specification of "frame"), laboratory procedures, and formulas for conversion of measurements.

SPECIES PROBLEM. Edited by Ernst Mayr. ix + 395 pp. American Association for the Advancement of Science, Washington, D. C., 1957. $8.75. The problem of species is relevant to many branches of biology—systematics, genetics, ecology, but also physiology, behavioral sciences and applied biology. Consequently, in the present volume the Science of Species is approached by the seven contributors from several angles and on the basis of diverse materials. The editor contributed a stimulating final chapter on "Difficulties and Importance of the Biological Species Concept."

MEASUREMENT OF LEVELS OF LIVING, WITH SPECIAL REFERENCE TO JAMAICA. By C. A. Moser. 106 pp. Her Majesty's Stationery Office, London, 1957. 13s. 6d. A distinction has been made between *levels* and *standards* of living, the former referring to actual living conditions of a people, the latter to their aspirations or expectations. The author singled out for study four aspects of levels of living: education, nutrition, health and housing (with some consideration given to income and expenditure).

FEDERAL FUNDS FOR SCIENCE. By National Science Foundation. iii + 60 pp. U. S. Government Printing Office, Washington, D. C., 1957. $0.40. The report provides detailed information on the expenditures of Federal agencies engaged in scientific research and development. The size of this operation is indicated by the fact that in fiscal year 1957, 23 agencies of the Federal Government obligated over $3 billion for these purposes.

CHEMICAL DYNAMICS OF BONE MINERAL. By W. F. Neuman and Margaret W. Neuman. xi + 209 pp. University of Chicago Press, 1958. $5.00. Several disciplines contribute to the growing knowledge of bone. The present work is offered as a synthesis of current physicochemical and biochemical facts and views, with surface chemistry of the mineral substance of bone as the foundation.

CEREBRAL CORTEX OF MAN. By W. Penfield and T. Rasmussen. xv + 248 pp. Macmillan, New York, 1957. $7.00. Based on some 400 brain operations carried out under local anesthesia, with positive evidence derived principally from cortical stimulation and negative evidence from cortical ablation, the monograph deals with the role of the cortex in perception, motor mechanisms, vocalization and arrest of speech, memory and dreams.

MANUAL OF NUTRITION. 67 pp. Philosophical Library, New York, 1957. $3.50.

Introduction to the principles of human nutrition, prepared in Great Britain by the Scientific Adviser's Division of the Ministry of Agriculture, Fisheries and Food.

RHYTHMIC AND SYNTHETIC PROCESSES IN GROWTH. Edited by Dorothea Rudnick. vii + 217 pp. Princeton University Press, Princeton, 1957. $7.50. The proceedings of the 15th symposium of the Society for the Study of Growth and Development consist of three sections, dealing, respectively, with recent advances in tissue culture methods, cyclic activities, and problems of biochemical evolution with emphasis on photosynthesis.

ANIMAL BEHAVIOR. By John Paul Scott. xi + 281 pp. University of Chicago Press, 1958. $5.00. Rare are the textbooks that may be read with pleasure by the student and have appeal to readers outside the captive audiences of a classroom. This, the reviewer believes, is one of them. Implications for human psychology are not ignored but the development of general theories of behavior is considered as the principal goal of the science of animal behavior.

A CLASSIFIED BIBLIOGRAPHY OF GERONTOLOGY AND GERIATRICS. By Nathan W. Shock. xxviii + 525 pp. Stanford University Press, Stanford, Calif., 1957. $15.00. The supplement registers close to 16,000 publications in all segments of this broad field. Contains an index of authors and subjects.

TRENDS IN GERONTOLOGY. By Nathan W. Shock. viii + 214 pp. Stanford University Press, Stanford, Calif., 1957. $4.50. Second edition, surveying major developments since 1950. It brings together many facets of the study of ageing and provides key references with illustrative data. Of special interest are the chapters on "Trends in research" and "Research potentials."

NUCLEAR RADIATION IN FOOD AND AGRICULTURE. Edited by W. Ralph Singleton. xii + 379 pp. D. Van Nostrand, Princeton, N. J., 1958. $8.50. Readers of this journal may be particularly interested in Part V of this volume (one of the outgrowths of the Geneva Conference, August 1955, on Peaceful Uses of Atomic Energy), dealing with genetic and biological hazards of nuclear radiation. Two papers (p. 259, 272) consider implications to human biology.

NO AND YES. By René A. Spitz. xii + 170 pp. International Universities Press, New York, 1957. $4.00. The author is engaged in a broad study of the ontogenesis of the ego from a psychoanalytical viewpoint. This report on the beginnings of language is the 6th in a series of investigations of man's early development.

BODY WATER IN MAN. By Maurice B. Strauss. xix + 286 pp. Little, Brown and Company, Boston, 1957. $7.00. Synthesis of the evidence that man is provided with distinct mechanisms for the regulation of the amount as well as the composition of the body fluids (thirst and appetite controlled by the central nervous system, and kidneys functioning under the influence of circulatory, hormonal and possibly neurogenic factors).

Science in Progress. Edited by Hugh Taylor. xv + 253 pp. Yale University Press, New Haven, Conn., 1957. $6.50. Tenth in the series, this volume presents reports of prominent scientists in a variety of fields, including Vannevar Bush's thoughtful essay on "Science and Progress?", the detective story by K. P. Oakley and J. S. Weiner about the hoax of "Piltdown Man," and R. R. Williams' "Chemistry as a supplement to agriculture in meeting world food needs."

Psychological Research. By Benton J. Underwood. vii + 298 pp. Appleton-Century-Crofts, New York, 1957. $4.00. The book's purpose is to aid in training psychology students in scientific method, with emphasis on experimental research. It may be perused with profit by all persons interested in the methodological issues vital to contemporary study of behavior.

Scientific and Technical Translating and Other Aspects of the Language Problem. 282 pp. UNESCO, Paris, 1957. $4.00. This volume, published in the series entitled "Documentation and Terminology in Science," deals with selected aspects of international scientific communication, with emphasis on the translations. The appendixes, in addition to other materials, contain a bibliography for technical-language study.

A Glossary of Some Terms Used in the Objective Science of Behavior. By William S. Verplanck. viii + 42 pp. American Psychological Association, Washington, D. C., 1957. $1.00. Aimed principally at increasing mutual intelligibility between ethnologists (usually trained in zoology and studying the behavior of insects, fishes and birds rather than of other groups) and behaviorists (usually trained in psychology, emphasizing the study of learning, and building their theories often on the behavior of the white rat). Not irrelevant for the study of human behavior.

Strategy of the Genes. By C. H. Waddington. ix + 262 pp. Macmillan, New York, 1957. $4.00. Essays in theoretical biology, dealing with the nature of biological organization and the theory of evolution. Noted here on account of the synthesis of the point of view of developmental biology and population genetics.

Methodology of the Study of Ageing. Edited by G. E. W. Wolstenholme and Cecilia M. O'Connor. x + 202 pp. Little, Brown and Company, Boston, Mass., 1957. $6.50. This volume is the third in the outstanding Ciba Foundation Colloquia on Ageing. It covers many approaches and facets of gerontology, from the study of cells and tissues to the consideration of whole organisms; from data on the egg production in hens and rattlesnakes to changes in human performance with age. Presentation of papers is followed by extended discussions. Changes in body composition were not considered.

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INFORMATION FOR CONTRIBUTORS

HUMAN BIOLOGY, a record of research, is a quarterly journal devoted to the subject its name implies. It publishes original articles dealing with human genetics, development, growth, reproduction and aging, functional anatomy, bio-anthropology, human ecology, population dynamics, etc.

The original manuscripts, typed double-spaced on one side of 8½ x 11 inch paper in complete and finished form *precisely as they are to appear*, should be sent—packed flat—*to the editor, Dr. Gabriel Lasker, Wayne State University College of Medicine, 1401 Rivard Street, Detroit 7, Michigan*. It is desirable that a second copy be included in order to expedite prompt consideration, and that an additional carbon copy and copies of all illustrations should be retained by the author.

Where possible, illustrations should be made for reproduction as line engravings, hence drawn in India ink as boldly as possible. Lettering must remain legible after figures are reduced to fit on 4¼ x 6¾ inch pages (letters 1½ to 2 mm high after reduction are adequate). Figures should be numbered consecutively with Arabic numerals. Tables should be numbered independently from 1 up, and both figures and tables should have captions. When the amount of tabular and illustrative material is judged to be excessive or unusually expensive, authors may be requested to pay the excess cost. Galley proofs will be sent to the author and must be returned promptly; when the author makes substantial departures from copy, the cost will be charged to him. Three copies of an abstract (not to exceed 225 words) should be submitted when the proof is returned. One will be sent to the editor of *Biological Abstracts*.

References in the text to the literature should be made by using the author's name followed by the year of publication: "Studies of Garn and Saalberg ('53) . . ." At the end of the article, under the heading "LITERATURE CITED," full references with titles and inclusive page references should appear (double spaced) in alphabetical order of the authors' names: "GARN, S. M. AND J. H. SAALBERG 1953 Sex and age differences in the composition of the adult leg. Human Biol., *25:* 144-153."

Authors will receive 25 free reprints. Orders for additional reprints should be placed at the time proof is returned. It will not be possible to accept orders for reprints after the proof has been returned to the press.

Human Biology

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a record of research

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THE ESTIMATION OF DRY SKELETAL WEIGHT BY PHOTOMETRY OF ROENTGENOGRAMS

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THE measurement of body composition in living man has improved considerably in the last few years. Perhaps the most important new measurement is the estimation of body fat by x-ray and specific gravity techniques. These methods permit one to quantify the fat and the fat free portion of the body. The use of biochemical techniques has increased our knowledge of body composition by providing measurements such as total body water, extracellular fluid and plasma volume. One of the major obstacles in our present estimate of body composition is a quantification of skeletal weight. While biochemical methods are available to determine whether one's bone mineral content is increasing or decreasing, no accurate method exists for providing an estimate of the total amount of bone present in the human body. In addition, bone mineral changes cannot, at present, be judged relative to the amount of bone present at the beginning of a balance study.

An anatomical approach has been utilized to estimate the skeletal weight by Merz *et al.* ('56) who have shown that there is a relationship between the weight of the dry, fat free skeleton and stature, femur length, area of the shaft and area of the compact bone in the femur

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midshaft. However, the use of all these independent variables provides a prediction equation which has a standard error almost equal to the standard deviation of skeletal weight. This permits the prediction of only three effective categories of skeletal weight: light, average and heavy. An analysis of the independent variables mentioned above indicated that the most significant one was the area of compact bone in the midshaft of the femur. This is to be expected since Trotter ('54) has shown that of all the single bones in the skeleton, the femur is the best correlated to total skeletal weight. Presumably the area of compact bone in the midshaft has a reasonably high relationship to total femur weight.

Investigations by McFarland ('54) with the bone density computer at Pennsylvania State University had indicated that the apparatus might give accurate measurements of the amount of mineral present in a bone section which had been x-rayed in the proper manner with a special aluminum alloy wedge. This suggested to the present investigators that a more accurate prediction of skeletal weight could be derived using this technique. The present paper reports the progress to date in developing a method for predicting the dry skeletal weight of a man from x-rays of his upper arm and thigh.

The equipment and its operation

The bone density computer consists of a tracing microdensitometer, an integrating device, mass counters, and a data recording device. To determine the density of a bone, a roentgenogram of the bone and calibration wedge is placed in the film holder. The images of the bone and wedge are traced along a predetermined straight line called a trace path: the wedge along a line parallel to its length and the bone along any desired line. The light passing through the wedge during the tracing process strikes a photomultiplier tube, light energy is thereby translated into an electrical signal. The signal is a monotonically increasing function of the wedge slope which is constant. This signal, at any instant, represents the total mass of the minute portion of wedge which the light beam traverses at that instant. By the use of an electromechanical integrating device, these signals are summed and presented in the form of a number which is a function of the total amount of wedge traced.

Inasmuch as the physical properties (density, composition, dimension) of the wedge are known, the number which represents the total mass count of wedge traced is directly related to a known quantity of aluminum alloy.

The most critical function of the integrating device is the adaptation of the densitometer: 1.) to variation in roentgenographic technique (exposing and processing film) and 2.) to the non-linear response of x-ray film to changes in x-ray intensity. This adaptation is effected by assigning an increasing step-series of specific number values to increasing film densities. This procedure determines that, when the bone is being traced, a given film density will be interpreted as a certain mass of aluminum alloy. Finally, a total mass count is obtained from the bone trace by a summing process in terms of a total mass of aluminum alloy. This mass count may be interpreted as a bone mass, since both bone and wedge can be described in terms of atomic content and x-ray mass absorption coefficients. The tracings of a femur and humerus cross section are illustrated in figure 1.

To convert these readings to bone density, an estimate of the volume of the cross section traced must be made and the mass divided by the volume. For the purposes of this study, the mass was the most useful measurement. Therefore, only mass readings are reported and are designated as mass coefficients. A more detailed description of the concepts and construction of this machine are given by Brown and Birtley ('51).

Determination of the number of trace paths required to predict accurately the weight of a whole bone

Work with rat femurs indicated that when a section of bone is traced, the weight and mineral content of that section can be predicted with great accuracy (the *r* values were .98 or better) (Schraer and Schraer, '56). However, tracing all the possible one-millimeter wide paths on a bone such as the human femur would require many days of work. Therefore, it was desirable to determine the optimum number of paths, *i. e.*, the minimum number which will still give satisfactory correlation to dry bone weight. Eleven human femurs taken from an anatomical collection were dried to a constant weight at a temperature of 70° C. They were then weighed and x-rayed with the wedge at 55 KVP, 60 milliampere-seconds and a 40 inch focal film distance. No-screen film and cardboard holders were used. Beginning .5 cm from the distal end of the bone, a total of 21 trace paths were made between the distal end and the mid-shaft. After these trace paths were evaluated twice, the 42 mass coefficients for each femur were averaged and correlated to the total femur weight. Four paths were then selected from the 21 and correlated to the weight, and, in addition, the midshaft tracing alone was correlated

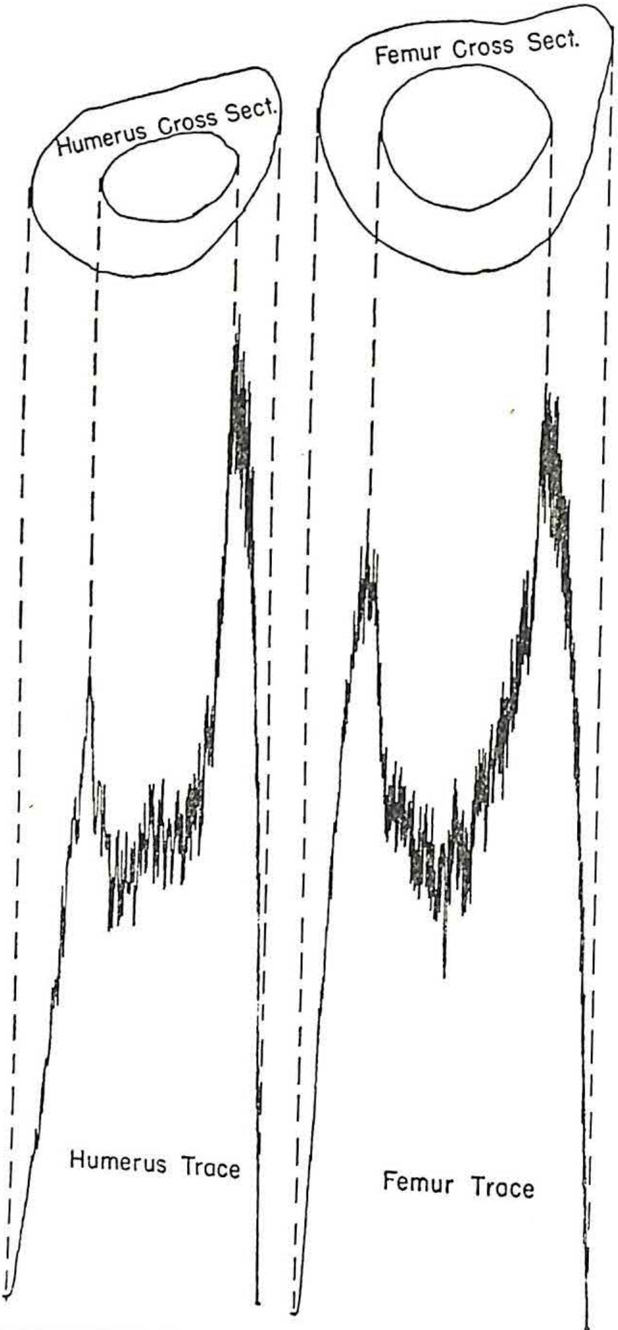

FIG. 1. DENSITOMETRIC TRACES OF A HUMERUS AND FEMUR THROUGH THE MIDSHAFT: SCALE OF FEMUR AND HUMERUS DIFFERENTIALLY EXPANDED FOR ILLUSTRATIVE PURPOSES. IN PRACTICE EACH TRACE IS RELATED TO THE WEDGE TRACE TAKEN FROM EACH FILM.

to weight. The relationship of the x-ray mass coefficients to the femur weight are shown graphically in figure 2. When twenty-one trace paths and four trace paths were used, the correlation coefficient (r) was .98 in both cases. This value was lowered to .94 when one trace path was used. Four trace paths on the distal half of the femur appear to be the maximum required for an accurate estimate of its weight. However, each trace on a femur requires about 15 minutes of machine time. Thus, one midsection trace path appears to provide sufficient information for routine evaluations, particularly when working with a group or with between-individual variations where maximum sensitivity is not required. When more accuracy is required four trace paths should be evaluated.

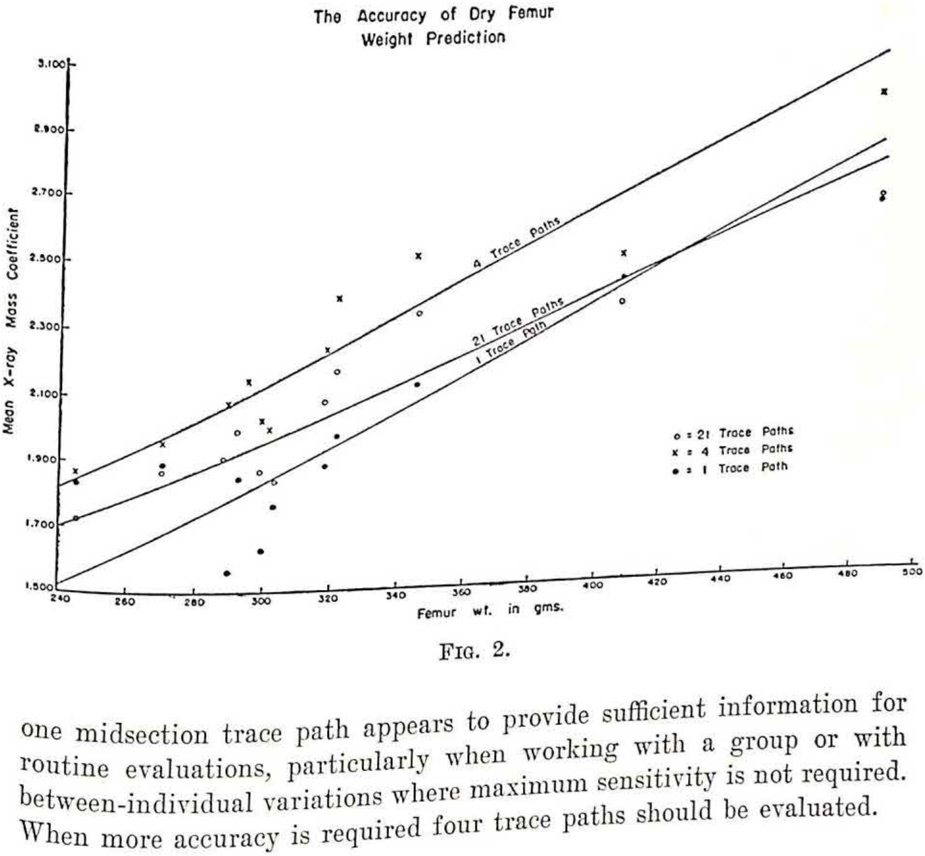

FIG. 2.

The relationship of femur and humerus x-ray mass coefficients to total dry skeletal weight

SAMPLE AND METHODS

The human skeletal material used to determine the relationship of x-ray mass coefficients to total dry skeletal weight consisted of Korean War Dead and has been previously described (Baker and Newman, '57). Along with the measurements described in the previous publication, roentgenograms of the right femur and humerus were made using the wedge. These plates were 8 by 10 Kodak no-screen film, enclosed in cardboard cassettes. The matched femur and humerus were placed with the anterior aspect facing the cassette and, after centering the x-ray tube 60 inches above the midpoint of the bones, the plates were exposed at 55 KVP for 324 milliampere-seconds. The midpoints on both bones were determined from the maximum length as measured on an osteometric board and were indicated on the x-ray plate with a lead arrow. These roentgenograms were processed through the bone density computer and, using the method previously described, a transverse tracing through the midsection of both bones was made. This process was repeated and the x-ray mass coefficients reported in this paper are the average obtained for these two tracings. After the elimination of poorly exposed films and unidentified cases, a total of 78 individuals were available for analysis.

RESULTS

An analysis of the component skeletal parts, as reported in the previous paper, indicated two significant factors which produced variation in the bone weights of the Korean War Dead material. These were the manner of burial and race. For this study only the Prisoner of War Dead material was used, and negro and white skeletal material was analyzed separately. The basic demographic and derived statistics on the material are summarized in table 1.

TABLE 1

Demographic and derived statistics on the populations

	WHITE $N = 64$		NEGRO $N = 17$	
	MEAN	S. D.	MEAN	S. D.
Age (yrs/mos)	25/5	6/2	26/4	3/8
Body Weight (kgs)	70.67	12.66	65.90	6.66
Height (cms)	173.6	6.6	173.9	5.9
Dry Skeletal Weight (gms)	4258.7	615.6	4583.0	441.6
Dry Femur Weight (gms)	409.2	62.5	423.9	41.6
Dry Humerus Weight (gms)	152.4	23.8	168.1	20.2
Femur Length (cms)	47.0	2.4	48.1	2.0
Humerus Length (cms)	33.5	1.7	33.8	1.7
Femur X-ray Mass Coefficient	2.519	.320	2.462	.194
Humerus X-ray Mass Coefficient	1.435	.226	1.512	.177

The femur and humerus were chosen for roentgenographic analysis for two reasons: first, they form a large portion of total skeletal weight and were shown by Trotter to be highly correlated to total dry skeletal weight ('54); second, because of their geometry they are fairly simple sites for densitometric analysis. As shown in table 2 the simple correlations of femur and humerus weight to total dry skeletal weight were not as high as those reported by Trotter. This may be due to the difference between burial and anatomical collection material. However, if the weight of both bones is correlated to the total skeletal weight by a multiple correlation, adequate prediction equations may be derived. Consequently, if the weight of these bones is accurately predicted, a usable estimate of total dry skeletal weight may be derived.

TABLE 2

The correlation of femur and humerus weights to total dry skeletal weight

	WHITES		NEGROES	
	ZERO ORDER r	MULTIPLE R	ZERO ORDER r	MULTIPLE R
Humerus Weight	.84	.93	.61	.83
Femur Weight	.89		.83	

The correlation of the x-ray mass coefficient of the individual bone to its weight was not as high as the preliminary tests on the anatomical series had suggested it would be. The femur mass to weight correlation coefficients were .81 for whites and .80 for the negroes. The humerus mass to weight correlation coefficients were .85 for whites and .74 for negroes.

In attempting to predict the total dry skeletal weight the bone lengths were also considered, as it was found by Merz *et al.* ('56) that these lengths sometimes improved prediction. Table 3 shows the simple and multiple correlation values which were obtained. The individual plots of x-ray mass coefficients in relationship to total dry skeletal weight are shown in figures 3 and 4.

In this sample the correlations indicate that the x-ray mass coefficients appear to be adequate predictors of total dry skeletal weight. The addition of femoral and humeral lengths do not substantially improve the multiple correlation coefficients for either the whites or the negroes. The lower multiple correlation coefficients found for the negroes are probably a product of the very limited range of bone weights encountered in this small sample. However, the samples could not be combined, as the negro and white regression slopes for bone weight to skeletal weight were divergent. This may be explained by the higher skeletal weight of the negroes and the differing x-ray mass coefficients. The samples showed that the negroes as compared to whites had a higher mean x-ray mass coefficient for the humerus, while they had a lower mean value for the femur.

TABLE 3

The intercorrelations of the predictive factors and their multiple correlation to total dry skeletal weight

			WHITES			
	HUMERUS X-RAY MASS COEFFICIENT	FEMUR LENGTH	HUMERUS LENGTH	TOTAL DRY SKELETAL WEIGHT	MULTIPLE CORRELATION TO DRY SKELETAL WEIGHT	
Femur X-ray Mass Coefficient	.548	.316	.330	.775	.862	.887
Humerus X-ray Mass Coefficient		.079	.043	.735		
Femur Length			.882	.362		
Humerus Length				.360		

			NEGROES			
	HUMERUS X-RAY MASS COEFFICIENT	FEMUR LENGTH	HUMERUS LENGTH	TOTAL DRY SKELETAL WEIGHT	MULTIPLE CORRELATION TO DRY SKELETAL WEIGHT	
Femur X-ray Mass Coefficient	.165	.596	.105	.603	.716	.729
Humerus X-ray Mass Coefficient		—.209	—.199	.479		
Femur Length			.679	.291		
Humerus Length				.108		

The regression equations for predicting total dry skeletal weight from various characteristics of the femur and humerus are given in table 4.

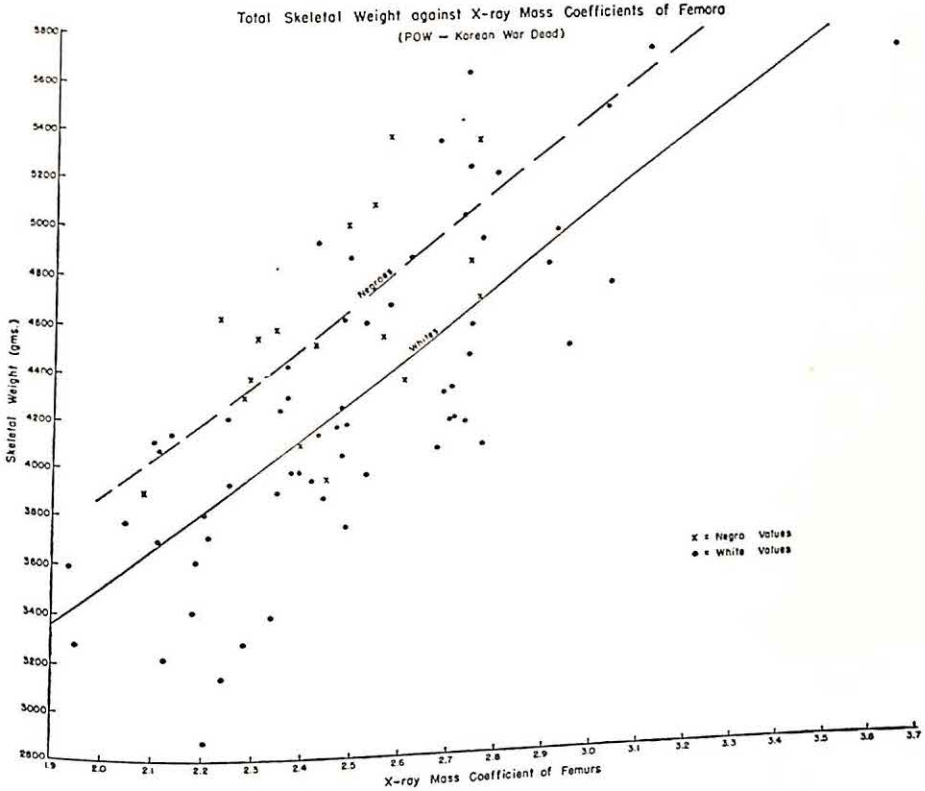

FIG. 3.

TABLE 4

Regression equations for predicting total dry skeletal weights

WHITES

Total dry skeletal weight = 2106.3 humerus x-ray mass coef. + 1272.8 ± 401.0
Total dry skeletal weight = 1567.6 femur x-ray mass coef. + 345.7 ± 426.0
Total dry skeletal weight = 1057.6 femur x-ray mass coef. + 1267.6 humerus x-ray mass coef. — 188.2 ± 323.9
Total dry skeletal weight = 2174.9 + 871.6 femur x-ray mass coef. + 1320.8 humerus x-ray mass coef. + 1514 femur lg. + 4.937 humerus lg. ± 295.3

NEGROES

Total dry skeletal weight = 1181.2 humerus x-ray mass coef. + 2812.9 ± 378.1
Total dry skeletal weight = 1346.6 femur x-ray mass coef. + 1283.5 ± 344.0
Total dry skeletal weight = 1191.8 femur x-ray mass coef. + 947.3 humerus x-ray mass coef. + 232.7 ± 301.7
Total dry skeletal weight = — 900.9 + 1368.9 femur x-ray mass coef. + 917.9 humerus x-ray mass coef. — 2.251 femur lg. + 5.182 humerus lg. ± 302.1

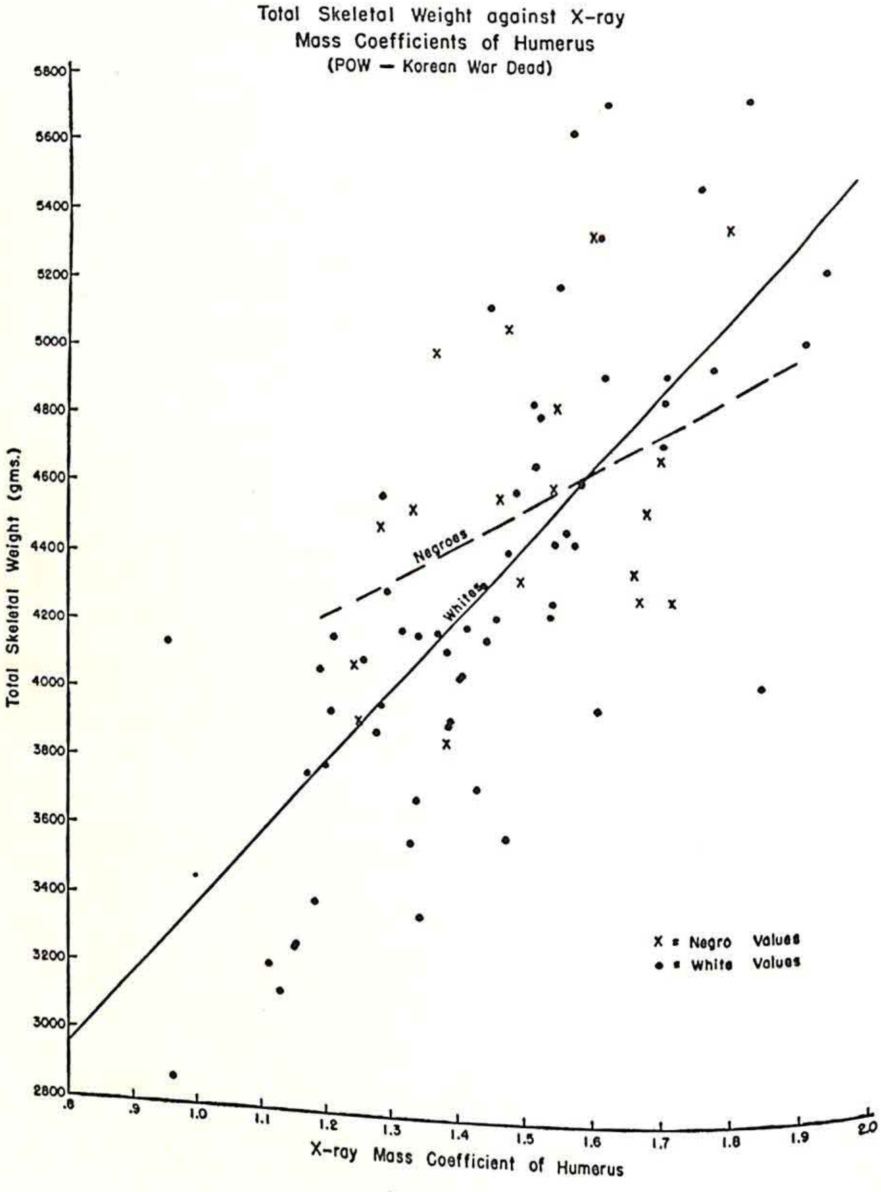
Total Skeletal Weight against X-ray
Mass Coefficients of Humerus
(POW — Korean War Dead)
Total Skeletal Weight (gms.)
5800
5600
5400
5200
5000
4800
4600
4400
4200
4000
3800
3600
3400
3200
3000
2800
.8
.9
1.0
1.1
1.2
1.3
1.4
1.5
1.6
1.7
1.8
1.9
2.0
X-ray Mass Coefficient of Humerus
Negroes
Whites
X = Negro Values
● = White Values

FIG. 4.

Although the correlation coefficients for the whites were much higher, it will be noted that the standard error of the predicted skeletal weight has approximately the same absolute value for both racial groups, again suggesting that the negro multiple correlation (R) values were governed by the small range of the skeletal weights. This low prediction error also hints that the regression equations for predicting Negro total dry skeletal weights are possibly as accurate as those derived for the whites. However, the number of cases used is so small that the extrapolation from this sample would be tentative, at best. For the whites, if the formulae including both x-ray mass coefficients are used, the standard error of the prediction is less than half of the standard deviation of skeletal weights. A slight improvement is gained by including the lengths. However, the improvement is small, and as the total length of these bones on the living is a difficult and inaccurate measurement, it seems better to use only the x-ray mass coefficients.

DISCUSSION

The present technique for estimating skeletal weight from roentgenograms is adequate for dry skeletal material provided the individual was between 18 and 40 years of age, and male. Furthermore, it is apparently better for whites than Negroes. Merz *et al.* ('56) have shown that it would probably not be safe to extrapolate these findings to females and, while no serious error would be encountered by extrapolating to older individuals, the equations will not apply to younger people.

In order to apply this technique to living subjects it would be necessary to correct for soft tissue encasing the bones. The bone density computing machine may be used for making a reading on the mass of the tissue surrounding the bone, and by appropriate calculations a correction could be made to the formulae as they now exist and the results applied to a direct estimate of total dry skeletal weight from roentgenograms of the living man. Two sources of error would not be corrected by this technique: first, the water content and certain organic constituents inside the living bone, which were not present in dry defatted bone, may produce an error and, second, it has been pointed out that large bones and large masses of soft tissue may produce considerable scattering error (Jackson, '51).

With the construction of a scintillation counter for the measurement of bone density (Mayer, '56), Trostle was able to make a direct measurement of the scattered radiation using various human extremity bones with, and without, ground meat surrounding them to simulate soft tissue ('57). From these studies Trostle concluded that the scattering error in measuring x-ray mass for a human extremity is less than 10% and is probably in the range of 3 to 8%; the scattering error increases slowly as the size of the object increases, and the soft parts produce most of the scattering error.

The only direct experimental evidence is the rat work reported by Schraer and Schraer ('57), who found that the femur x-ray mass readings from x-ray plates of living rats correlated .98 to the values on plates from the excised femurs.

It thus appears that a direct comparison of x-ray plates on intact and excised human femora and humeri will be required before we can be certain of the present correction techniques. A series of plates on cadavers is now being made and, when complete, should provide us with both the exact error factor related to soft tissue and a correction for any systematic error involved in the present soft tissue correction.

Trotter and Peterson ('55) have shown that for most non-pathological individuals the ashed weight of the skeleton formed 64.8% to 66.9% of the dry skeletal weight. However, they also found one skeleton where the ashed weight was only 54% of the dry skeletal weight. If this individual did not during his lifetime have some disorder that affected his bone mineral content, we must asume that dry bone weight is not always a close predictor of bone mineral even for non-pathological individuals. Work with x-ray mass coefficients for rat femur has shown that these values reflect bone ash content as closely as dry weight (Schraer and Schraer, '56). Thus, x-ray mass coefficients for human femora and humeri may be better indicators of the total bone mineral than the predictions of dry skeletal weight indicate, but the work of Trotter and Peterson ('55) also indicate that shifts in bone mineral are more common in other bones than the femur and humerus. Therefore, it appears that direct experimental work will be required before we can determine how accurately our present techniques will predict total bone ash in the body.

The presence of any pathological condition which affects bone would probably change not only the relationship of one skeletal component to another, but also the relative mass of different parts of the same bone. Thus, the present formula would probably not be accurate for any individual with an osseous disorder. Because of these limitations, we visualize this technique to have two major applications: first, it may offer an independent measure of bone weight for correcting and improving measurements of body compositions; and second, it may provide a tool for investigation of the effects of nutriture and genetics on the growth and development of the skeleton.

SUMMARY

Methods of measuring body composition have improved considerably in the past few years. However, present techniques do not give any accurate method of measuring total dry skeletal weight or total bone mineral in the living man. In the present study we have shown how a reasonably accurate estimate of total dry skeletal weight may be made from roentgenograms of the humerus and femur of adult males. The material consisted of: the dry weight and x-ray plates of 11 femora from an anatomical collection; the dry, defatted total skeletal and individual bone weights of 61 whites and 17 Negroes who died in the Korean War plus x-ray plates of their right femur and right humerus. An aluminum alloy wedge which served as a standard was exposed simultaneously with the bones. The films were evaluated in the bone density computing machine, which gives a value of the mass in terms of aluminum alloy for the section of the x-ray film studied and corrects this reading for differences in the film due to variations in exposure and development.

Experiments with the 11 femora from the anatomical collection indicated that a single trace path across the midshaft was adequate to predict total femur weight. The Korean War Dead material showed that the weights of the femora and humeri would accurately predict total skeletal weight for the whites and, with limited accuracy, for Negroes. An evaluation of the x-ray film though the midshaft of these bones yielded x-ray mass coefficients which predicted the individuals' total dry skeletal weight with a standard error equal to approximately one half of the standard deviation of dry skeletal weight. The addition of bone lengths did not significantly improve the multiple correlations. These results were obtained on skeletal material and cannot be applied to the living without further modification of the technique or the derivation of a soft parts correction. The development of this correction is in progress and, if completed successfully, the estimation of dry skeletal weight should offer a valuable adjunct to our methods of measuring normal human body composition.

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SOME MONTE CARLO METHODS APPLIED TO AN EPIDEMIC OF ACUTE RESPIRATORY DISEASE

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INTRODUCTION

FOR over half a century attempts have been made to approximate the course of epidemics by the use of mathematical models. In the pioneer work on this subject many aspects of the transmission of disease were considered and many assumptions were made about such things as the virulence of organisms, susceptibility of populations, periods of incubation, and so on. The resulting epidemic curves, usually expressed by the weekly incidence of new cases, were reproduced in a wide variety of situations. Generally speaking, this early work was deterministic in nature, ignoring variation in epidemics and being applicable primarily to large populations. Reference is made to the fine review article by Serfling ('52).

Following the preliminary attempts at mathematical model building, developments by many investigators, including Greenwood ('20, '31, '36), Feller ('39, '43) and Lundberg ('40), drew attention to the use of stochastic process theory and probabilistic models in this field. As a consequence, during the past 15 years attention has been turned with increasing frequency to the study of epidemic phenomena by probabilistic methods. A series of papers by Bartlett ('49, '53, '56), Kendall ('48, '50), Bailey ('50, '53, '55, '56) and Whittle ('52, '55) report studies which were made of several simple epidemic models. In addition to information on the course of an epidemic, results have been obtained on the total size of an epidemic, on chain-type situations, on recurrent epidemics, and on threshold conditions leading to major or minor epidemics. One discouraging result has been the exceedingly complicated mathematical problems which come from incorporationg necessary conditions into the models. For the past 3 or 4 years a few experimenters have attempted to avoid these complications by using random sampling or Monte Carlo techniques. Recent work by Bartlett ('57) and Kendall ('56) illustrates this.

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* Now at The School of Public Health, University of California, Berkeley.

The present paper presents a probabilistic model and its deterministic counterpart which were developed to describe the course of acute respiratory disease among recruits at a former Air Force reception center at Sampson Air Force Base. Four parameters are estimated in order to fit the model to actual data, and the model is then used to create several hundred artificial epidemics. The frequency distributions of the numbers of cases per week so obtained are compared with actual observations. While patterned after the classical models studied by Bartlett, Bailey, and Kendall, this work brings in two complications not present in early models. Partial immunity and subclinical cases are admitted as possibilties and their properties are determined by fitting the model to the observations.

Epidemic situation

During the winter of 1952-53 at Sampson Air Force Base in upper New York State observations were made of the incidence of upper respiratory disease among a large number of young men newly recruited by the Air Force. See Bernstein ('57) for further description of the base. As men entered the Air Force in the eastern part of the United States they were sent immediately to this base. Upon arrival they were organized into groups, called "flights," most of which were composed of either 60 or 72 men. For 12 weeks thereafter, they lived, ate, and worked together in these flights. There was considerable cohesiveness within these groups and the members of a flight tended to stay together On the other hand, there was a mingling of men from all over the base at theaters, games, stores, and so forth. In terms of exposure, each man was exposed to infection from infected men in his own flight as well as to an additional, fairly continuous level of infection existing within the base environment.

All flights of recruits assigned to areas G and H of Sampson Air Force Base between November 1, 1952 and March 1, 1953, and consisting of 60 men each, were chosen for study in this paper. There were 117 of these and they were observed throughout the entire 12 weeks of training. Whenever a man was hospitalized for upper respiratory disease the diagnosis was recorded. Only those diagnoses which were labeled "acute respiratory disease" (ARD) were included in this work.

ARD was defined, Bernstein ('57), as "acute illnesses with respiratory symptoms or constitutional reactions, or both, in which there was no increase in the influenza antibodies, a negative throat culture for group A streptococci, and hospitalization for less than seven days." In recent years members of a certain family of viruses called "adenoviruses" have been established as causative agents accounting for a sizeable portion of cases of ARD, Hilleman ('54, '55) and Rowe ('53, '56). Experiments using volunteer subjects, Commission on Acute Respiratory Diseases ('47), revealed that the incubation period is about 5 to 6 days, wtih fever lasting 2 to 4 days and some symptoms persisting for two weeks. While there is evidence that infection by a specific type of this virus family results in immunity to that type, such immunity does not seem to hold for other types.

A study, Hilleman ('55), was made in 1954 at an Army base in which evidence concerning infection wtih one of the above virus types (RI 67) was obtained on a large number of recruits. It was found that over 80% showed signs of having become infected during their first four months in the Army. Of these, only about 25% were hospitalized, indicating that many cases of ARD are subclinical and consequently not observed.

Table 1 presents the number of observed cases; *i.e.*, hospitalizations, by week, for the 117 flights observed. The pattern shown by the trend of weekly hospitalizations is typical of military recruit training centers with its peak at the fourth week and a rapid decrease beyond that peak. Three things are of particular interest: 1. There must have been some sort of immunity developing, because of the fall in number of cases after the fourth week. This decrease occurred in spite of a general and uniformly high level of incidence over the entire base during the period of the study. 2. There were many second and a few third cases, indicating that the immunity developed was not complete. 3. Apparently only a small proportion of the population was affected. There were 1,476 hospitalized out of 7,020 men. From observations made at the Army base mentioned above, it seems probable that there was a large amount of unapparent infection. These considerations formed the basis of attempts at constructing a model.

The distribution of the number of cases occurring in any one week, as shown in table 1, is interesting in itself. During the first week this distribution cannot be distinguished from a binomial. However, most of the following weeks show a variation much greater than would be expected from a binomial situation, which is in line with what one would expect from a process in which contagion exists.

TABLE 1

Number of flights by number of first and number of repeat hospitalizations for ARD by week at Sampson AFB among 117 flights arriving between November 1, 1952 and March 1, 1953

No. of first hospitalizations	WEEK AT SAMPSON 1	2	3	4	5	6	7	8	9	10	11	12
0	94	42	16	12	22	56	78	97	99	111	103	97
1	18	30	30	16	27	30	29	14	14	6	13	15
2	5	24	19	22	24	15	9	3	2		1	4
3		10	15	14	17	9	1	1	2			1
4		6	12	17	11	3		1				
5		3	7	11	4	4						
6		1	6	10	4			1				
7		1	4	7	6							
8			4	3	2							
9			3	3								
10												
11				2								
over 11			1 *									
Total number	28	160	336	407	272	119	50	33	24	6	15	26
Mean per flt.	.24	1.37	2.87	3.48	2.32	1.02	.43	.28	.21	.05	.13	.22
Std. Dev.	.52	1.48	2.70	2.53	2.05	1.31	.67	.82	.30	.22	.36	.54
No. of repeat hospitalizations												
0	117	117	115	105	102	101	110	110	109	112	110	106
1			2	8	13	16	6	7	8	4	7	10
2				4	2		1			1		1
Total number			2	16	17	16	8	7	8	6	7	12
Mean per flt.			.017	.137	.145	.137	.068	.060	.068	.051	.060	.103

* In one flight 17 cases were observed in the third week.

Mathematical model

The primary purpose of this paper was to construct a model epidemic with properties which were compatible with the epidemic situation and observations. Consequently, a model was built with the following properties.

1. The probability of a highly susceptible man becoming infected from outside the flight in a small interval of time Δt is $\mu \Delta t + o(\Delta t)$, where μ is a constant. It follows that the probability α of a man escaping infection from outside in a unit period of time is $\alpha = e^{-\mu}$.

2. The probability of infection from within the flight in a small interval of time Δt is dependent upon I the number of infectious men within the flight. This probability is assumed to be $\lambda I \Delta t + o(\Delta t)$, and it follows that the probability of escaping such infection during a unit period of time, during which I is constant, is

$$\beta^{I} = e^{-\lambda I}.$$

3. Once recovered from infection, a man has partial immunity, and the constants μ and λ in 1 and 2 above are both altered by multiplying them by the same factor γ.

4. There exists a proportion, δ, of men who become infected but are not detected. These men remain in the group and provide the pool of infection from within.

5. Incubation periods are about 5 days.

6. Periods of infectiousness are unknown but are assumed to be less than 7 days.

In constructing a model based on these properties the 12 weeks of observation were considered separately. During each week the risks of infection were assumed to be constant, depending on the occurrences of the previous week. Remembering that only men placed in the hospital were observed, the following states were postulated. They account for the observations under the properties of the model.

State 1. Highly susceptible. No previous infection.

State 2. Moderately susceptible. One or more previous infections. Never hospitalized.

State 3. Moderately susceptible. At least one previous hospitalization. (Note: States 2 and 3 must be kept separate in order to identify men in States 8 and 9 below.)

State 4. Infectious. An unobserved case infected for the first time.

State 5. Infectious. An unobserved case infected at least once before. Never hospitalized.

State 6. Infectious. At least one previous hospitalization.

State 7. Hospitalized for the first time, no previous infection.

State 8. Hospitalized for the first time, one or more previous infections.

State 9. Hospitalized. At least one previous hospitalization.

The total number of men in States 7 and 8 were observed as first hospitalization. State 9 was observed as repeat hospitalizations. The remainder of the states were not observed but might have been under proper circumstances. Men in States 4, 5, and 6 provide the pool of infection from within the flight. This total is called I in property 1 above.

Some further assumptions were made. Actually a man can become infected at any time. In the model it was assumed that any changes occur at the close of each week. At that time infected men either enter the hospital or become and remain infectious and undetected. At the end of the following week they are well again and become moderately susceptible.

Table 2 presents the probabilities of changing from one state at time t to another state at time $t+1$. These are called transition probabilties. Justification for their choice follows. Suppose at time 0 all 60 men of the flight are well and highly susceptible (State 1). At the end of the first week the probability of a man remaining well (*i.e.*, of remaining in State 1) is the probability α of escaping infection from without.

$$\alpha = e^{-\mu},$$

where one week is the unit of time. The probability of becoming infectious and undetected (State 4) is $\delta(1-\alpha)$ and that of going to the hospital (State 7) is $(1-\delta)(1-\alpha)$, as implied by property 4.

Assuming that the close of the first week finds N_{11} men in State 1, N_{14} in State 4, and N_{17} in State 7, the probability of a man in State 1 remaining well during the second week is now

$$\alpha\beta^{I_1} = e^{-\mu}e^{-\lambda I_1}$$

where I_1 equals N_{14} which is the number of infectious men present at time $t=1$. The probability of going from State 1 to State 4 is now $\delta(1-\alpha\beta^{I_1})$, and that of going from State 1 to State 7 is $(1-\delta)(1-\alpha\beta^{I_1})$.

TABLE 2

Transition probabilities from time t to time $t+1$

	STATE AT TIME t	STATE AT TIME $t+1$								
		SUSCEPTIBLE			INFECTIOUS			HOSPITALIZED		
		1	2	3	4	5	6	7	8	9
SUSCEPTIBLE	1 Highly susceptible (no previous case)	$\alpha\beta^{I_t}$	0	0	$\delta(1-\alpha\beta^{I_t})$	0	0	$(1-\delta)(1-\alpha\beta^{I_t})$	0	0
	2 Moderately susc. (no prev. hosp.)	0	$\alpha^{\gamma}\beta^{\gamma I_t}$	0	0	$\delta(1-\alpha^{\gamma}\beta^{\gamma I_t})$	0	0	$(1-\delta)(1-\alpha^{\gamma}\beta^{\gamma I_t})$	0
	3 Moderately susc. (one or more hosp.)	0	0	$\alpha^{\gamma}\beta^{\gamma I_t}$	0	0	$\delta(1-\alpha^{\gamma}\beta^{\gamma I_t})$	0	0	$(1-\delta)(1-\alpha^{\gamma}\beta^{\gamma I_t})$
INFECTIOUS	4 Infectious (no previous case)		1							
	5 Infectious (no prev. hosp.)		1							
	6 Infectious (one or more hosp.)			1						
HOSPITALIZED	7 Hospitalized (no previous case)			1						
	8 Hospitalized (prev. case but no prev. hosp.)			1						
	9 Hospitalized (one or more prev. hosp.)			1						

At the close of the second week the men in State 4 all enter State 2 and those in State 7 enter State 3 in accordance with the assumptions above.

Once infected and recovered (States 2 and 3) a man has partial immunity. In this situation the probability of remaining well during the $(t+1)^{\text{th}}$ week is

$$\alpha^{\gamma}\beta^{\gamma I_t} = e^{-\gamma\mu}e^{-\gamma\lambda I_t}$$

where γ is an index of immunity as given in property 3 above. A man in State 2 can remain there or go to States 5 or 8. A man in State 3 can remain there or go to States 6 or 9. All these transitions are summarized in table 2.

The parameters introduced above have fairly obvious meanings. The probability of escaping infection from outside is α. A contagion factor, β, is utilized once infections occur in a flight. Obviously, both α and β are between 0 and 1. $\beta = 0$ implies overwhelming contagion, and $\beta = 1$ implies no contagion. δ is the probability that, given a man is infected, he is not hospitalized. $\delta = 0$ corresponds to a situation in which everyone is isolated when infected. $\delta = 1$ means no infected people are isolated. In this model δ is assumed constant, regardless of the number of previous infections. γ is an immunity index. $\gamma = 0$ implies perfect immunity, $\gamma = 1$ means no immunity, and $\gamma > 1$ implies an increased susceptibility due to earlier infection.

Estimation of parameters

The problem of estimating the 4 parameters in this model is not simple and the project would have been abandoned without the presence of a CRC 102A electronic computer obtained from the National Cash Register Company. The method of choice in this estimation was maximum likelihood, but unfortunately the complexity of the model was such that the use of this method seemed impossible. Consequently, other (more questionable) means were adopted. A deterministic counterpart of the model was used to obtain expected values corresponding to the observations of first and repeat hospitalizations. Given values for α, β, γ, and δ, the transition probabilities were completely determined. Then, beginning with 60 men in State 1 in the first week, the expected numbers of men in States 1, 4, and 7 in the second week were computed. These in turn were used to get the various expected numbers for the third week. Thus for a given set of parameters which determined the transition probabilities, the deterministic counterpart could be computed week by week. The computations were programmed for the electronic computer so that a complete deterministic epidemic for a flight of 60 men could be produced. The usual χ^2 goodness of fit expression was then used, not in the ordinary sense but merely as an index of the deviation of deterministic values from the observations. The computation of this index was also programmed for the computer and eventually a set of parameters could be inserted into the model, the deterministic results computed, the χ^2 index computed, and the index typed out on a flexowriter in two and a half minutes. This formed the

TABLE 3

Number of flights by number of first and number of repeat hospitalizations by week of exposure of 415 Monte Carlo flights to a model of an epidemic of ARD

No. of first hospitalizations *	WEEK OF EXPOSURE 1	2	3	4	5	6	7	8	9	10	11	12
0	263	132	51	23	54	112	225	259	311	336	347	370
1	122	126	74	55	89	148	120	121	90	68	60	42
2	26	81	93	94	111	93	50	31	14	10	8	3
3	3	53	73	81	76	42	13	3				
4	1	17	47	76	48	15	6			1		
5		6	34	43	21	5		1				
6			20	26	12		1					
7			14	9	2							
8			6	3	1							
9			3	4	1							
10				1								
Total number	187	545	1130	1294	939	545	289	197	118	92	76	48
Mean per flt.	.45	1.31	2.72	3.12	2.26	1.31	.70	.48	.28	.22	.18	.12
Std. dev.	.67	1.24	2.00	1.84	1.60	1.15	.94	.70	.52	.50	.43	.34

No. of repeat hospitalizations †	1	2	3	4	5	6	7	8	9	10	11	12
0	415	415	413	392	362	362	369	390	396	392	398	397
1			2	22	51	51	45	25	19	23	17	16
2				1	2	2	1					2
Total number			2	24	55	55	47	25	19	23	17	20
Mean per flt.			.005	.058	.133	.133	.113	.060	.046	.055	.041	.048

* Sum of numbers in states 7 and 8, see table 2.

† Number in state 9.

basic procedure for obtaining estimates of the four parameters, α, β, γ, and δ. A rapidly converging iterative process was found. Two parameters were guessed, and the other two were determined by trial and error to minimize χ^2. These two minimizing values were then fixed, and the first two parameters were varied to minimize the χ^2 once more. Six repetitions of this provided estimates accurate to three decimal places. Various guesses were tried as a starting point, all resulting in the same limiting estimates which were the following:

$$\alpha = .962 \qquad \gamma = .167$$
$$\beta = .941 \qquad \delta = .827.$$

The deterministic epidemic resulting from these estimates is given in table 4.

TABLE 4

Weekly incidence of hospitalizations for observed, Monte Carlo, and deterministic flights

	FIRST HOSPITALIZATION			REPEAT HOSPITALIZATION		
Week at Sampson AFB	117 observed flights	415 Monte Carlo flights(1)	Deterministic flights(2)	117 observed flights	415 Monte Carlo flights(1)*	Deterministic flights(2)†
1	28	53	46			
2	160	154	166			
3	336	319	368	2	1	1
4	407	365	418	16	7	6
5	272	265	243	17	16	17
6	119	154	121	16	16	18
7	50	81	70	8	13	13
8	33	56	44	7	7	10
9	24	33	31	8	5	7
10	6	26	24	6	6	6
11	15	21	20	7	5	5
12	26	14	18	12	6	5
All weeks	1476	1541	1569	99	82	88

* Incidence obtained for 415 flights multiplied by $\frac{117}{415}$.

† Deterministic mean multiplied by 117.

The value of χ^2 was approximately 72 when all 22 weekly observations of first and repeat hospitalizations were used in the fitting process. This was not a spectacularly good fit but as mentioned later the model certainly seems to have retained the features of the epidemic.

Monte Carlo epidemics

In accordance with the model as given in table 2, the transition probabilities depend on the parameters α, β, γ, and δ. Using the values obtained from the estimation just described, and assuming that 60 highly susceptible men begin in each flight, one can observe a "Monte Carlo flight" as it proceeds through 12 weeks of exposure. Such a flight was constructed by the following steps:

Step 1. First week. The computer generated 60 pseudo random numbers between zero and unity by a method analogous to those discussed in Cameron ('54), Juncosa ('53). Of these, the numbers which were less than α were taken to be men who remained well for the first week. Those between α and $\alpha + \delta(1 - \alpha)$ were men who received unapparent infections (these were put equal to I_1), while those greater than $\alpha + \delta(1 - \alpha)$ were men who were hospitalized. Suppose, for illustration, these groups had 57, 2, and 1 men in them, respectively.

Step 2. Second week. For the second week 57 random numbers were drawn, and those less than $\alpha\beta^{I_1}$, where $I_1 = 2$ in this example, remained well, those between $\alpha\beta^2$ and $\delta(1 - \alpha\beta^2)$ received unapparent infections, and those greater than $\delta(1 - \alpha\beta^2)$ were hospitalized. Now assume the observed numbers to be 52, 4, and 1, respectively.

Step 3. Third week. For the third week there were two men in State 2 and one in State 3 as a result of the first week's experience. Two random numbers were drawn and those less than $\alpha^{\gamma}\beta^{\gamma I_2}$, $(I_2 = 4)$, remained well in State 2 with the others being infected analogously to Step 2. Similarly, one random number was drawn yith the same disposition rule. The 52 remaining well men were handled as the 57 had been in Step 2.

Steps 4-12. Analogous to Step 3.

These steps were carried out for 12 weeks of exposure by the electronic computer. About 100 Monte Carlo flights could be exposed to the conditions of the model in 8 hours of machine time. A total of 415 flights were so exposed. Tables 3, 4, and 5 present some of the findings. Figure 1 is based on table 4. Comparing weekly hospitalizations for the observed and Monte Carlo flights by the χ^2 index gives a value of χ^2 equal to 98, somewhat larger than the value of 72 obtained by comparing the observed and deterministic results.

Table 3 presents the 415 Monte Carlo epidemics in a manner comparable with the presentation of the 117 epidemics in table 1. In table

TABLE 5

Percent of 117 observed flights and 415 Monte Carlo flights by number of hospitalizations for ARD during a 12 week period

NUMBER OF HOSPITALIZATIONS DURING 12 WEEK PERIOD	PERCENT OF FLIGHTS WITH x HOSPITALIZATIONS AS FOUND IN	
(x)	117 observed flights	415 Monte Carlo flights
0–4	1.7	.2
5–9	24.8	8.7
10–14	35.9	50.6
15–19	23.9	35.9
20–24	9.4	4.6
25–29	3.4	
30 and over	.9	
Mean *	13.5	13.8
Std. deviation *†	5.5	3.3

* Based on the data prior to grouping.

† There is a significant difference at the 0.01 level between the two standard deviations.

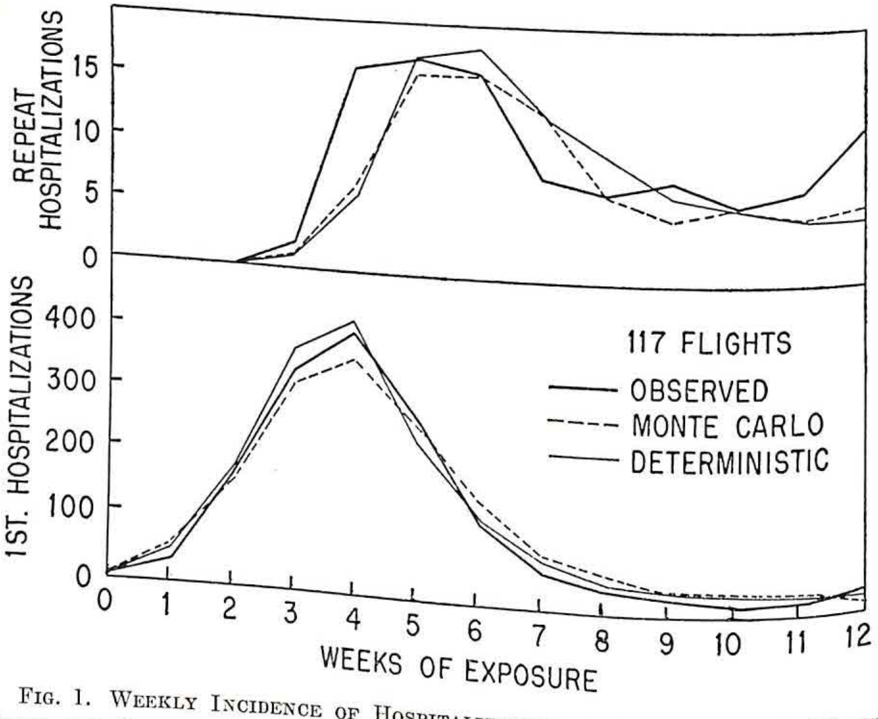

FIG. 1. WEEKLY INCIDENCE OF HOSPITALIZATIONS FOR 117 OBSERVED, MONTE CARLO, AND DETERMINISTIC FLIGHTS (See table 4 and its footnotes).

4 the weekly incidence of the observed, the Monte Carlo, and the deterministic epidemics are compared. Figure 1 presents these graphically. It appears that the model has come fairly close to the observations of first hospitalizations but that it cannot match the pecularities observed in repeat hospitalizations.

DISCUSSION

There are a number of questions associated with the epidemic model of this paper. First to be noticed is that this epidemic situation is a continuous process in time which has been forced to be a discrete one. Consequences of this are not clear, but are assumed to be slight. The incubation period of 3–5 days is shortened a bit in the model by assuming that men infected during one week are infectious during the following week. As a result of this assumption, the first week should show fewer observed cases than predicted by the model. This turned out to be true. The period of infectiousness for ARD is probably quite variable but the observations on volunteers mentioned earlier, Commission on Acute Respiratory Diseases ('47), indicate that the one week used in the model is a fairly reasonable estimate. The time a man is isolated in the hospital is assumed to be one week. This is probably too long, but unfortunately data were not available on this point.

More serious than the above is the fact that this model ignores men who were absent from the flight for reasons other than ARD. There was a lot of streptococcal disease and a sharp influenza outbreak during the period under study. Another difficulty is that men with extended absences were set back in their recruit training and transferred to other flights. Such transfers were not accounted for in this study. A third objection is that prior to being hospitalized infected men undoubtedly contributed to the risk of infection; however, the model ignores this.

The epidemic model of this paper, through its 4 parameters, has considerable versatility with respect to immunity, undetected cases, and contagion. Changing any of the parameters by as much as a few percent causes considerable change in the χ^2 index. The assumption that all men are equally prone to infection at time zero could have been modified in a simple manner by using a fifth parameter. However, the nearly binomial distribution observed for the first week lends credence to the equality of initial proneness which was assumed.

For simplicity, the model assumed that the risk of infection from without was constant. Examining hospitalization rates for the entirety of Sampson Air Force Base reveals that this assumption was only approximately true, there being a lower risk in November and March than in the intervening months. This may account in part for the finding that the observed flights had greater variability in weekly incidence than did the Monte Carlo ones.

If the model were basically correct, the estimates of the 4 parameters would indicate something of the nature of the disease. Since $\delta = .827$, we would say there is indication that a large proportion, over 80%, of infections are subclinical. We would also say that $\gamma = .167$ implies that recovery from an infection results in partial immunity which increases the probability of escaping infection from outside the flight in one week from .962 to $(.962)^{.167}$ or .994. There is danger in attaching too much physical significance to the values of these parameters, however. There is always the possibility that the model used, while fitting the data, is not unique and is not a true representation of the underlying mechanisms.

The probabilistic theory of epidemics has long been concerned with the distribution of subjects in various states at a given time. Only rarely, however, are enough data available to observe these distributions empirically and to compare them with theoretical ones. The types of data used in this work are somewhat rare. Studies of families constitute the main effort in this direction. Of course similar information has long been collected at military installations, but never, to the author's knowledge, has it been published or studied from the viewpoint of this paper. It provides a means of checking theoretical models and, once the mathematical problems of estimation and distribution theory are solved, may lead in the future to far-reaching conclusions concerning the housing and treatment of susceptible populations.

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CHANGING TRENDS IN THE CHINESE-AMERICAN POPULATION

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CHINESE immigration to the United States won considerable publicity during the last 4 decades of the nineteenth century as the volume of immigration grew. In recent years, in contrast, the Chinese population has received relatively scant attention. This lack of interest attests to the changed role of the Chinese in the American economy. Briefly, because of the elimination of the Chinese from jobs desired by the whites, the Chinese question lost its economic significance and tolerant indifference took the place of hostility (McWilliams, '43).

Along with this altered economic role, other changes also occurred. Geographically, wider dispersion of the Chinese rapidly took place. The exclusion laws passed in 1882 and in subsequent years prevented additional substantial immigration. As a result, the Chinese suffered a consistent decline for several decades. Parallel with the decrease in numbers was a gradual increase in average age. The ability of the Chinese to survive was thus in doubt (U. S. Senate, '11).

With possibly only one exception (Thompson and Whelpton, '33), doubt as to the future of the Chinese population has persisted to the present. Lee ('47) predicted that "the Chinese population in this country is likely to decrease rather than increase." King ('50) expressed a similar, though less pessimistic, belief that "there would be some increase in the Chinese-American population in the next decade or so (1940-1950). Probably, however, no substantial increase or decrease will be observed."

In the present paper, this pessimistic opinion is assessed quantitatively in terms of the sex and age characteristics of the Chinese population. Attention will be particularly focused upon more recent development insofar as the data permit.

In retrospect

The number of Chinese arrivals in the United States up to 1850 was variously estimated (cf. Coolidge, '09; Ferenczi, '29). According to one source, only 46 Chinese were recorded arriving in continental United States between 1820 and 1850. Of the 46, 31 were males and 15 females (Bromwell, 1856). The census of 1850 gave, without distinction of sex, a total of 758 Chinese in the country as a whole. Notwithstanding the discrepancies, the number of Chinese in this country could not have been very large in 1850.

TABLE 1

Nativity of the Chinese population in the United States, by sex, 1850-1950

		Male			Female		
Year	Grand total	Total	Foreign born	American born	Total	Foreign born	American born
1950	117,140	76,725	40,469	36,256	40,415	14,581	25,834
1940	77,504	57,389	31,687	25,702	20,115	5,555	14,560
1930	74,954	59,902	39,109	20,693	15,152	4,977	10,175
1920	61,639	53,891	40,573	13,318	7,749	2,534	5,214
1910	71,531	66,856	54,935	11,921	4,675	1,661	3,014
1900	89,863	86,341	78,684	6,657	4,522	2,169	2,353
1890	107,488	103,620	*	*	3,838	*	*
1880	105,465	100,686	100,038	648	4,779	4,244	535
1870	63,199	58,633	58,325	308	4,566	4,357	209
1860	34,933	33,149	*	*	1,784	*	*
1850	758	*	*	*	*	*	*

Source: United States censuses. * No separate figures available.

Table 1 clearly reflects the great influx of Chinese in the 4 decades after 1850. The total number of Chinese would undoubtedly have been many times greater had not many of them returned home before their presence was recorded in the census.

Evident also in table 1 is the effect of the passage of the Exclusion Laws. With the greatly reduced immigration after 1882, the total number of Chinese dropped from 107,488 in 1890 to 61,639 in 1920. It was not without reason that the Immigration Commission in 1911 (U. S. Senate, '11) questioned the ability of the Chinese to survive.

However, as table 1 further suggests, the failure of the Commission to take into account the growing number of American-born Chinese in the population rendered its conclusions invalid.

The total number of Chinese rose to 117,140 in 1950. Except for 1930 and 1950 when increments in the number of foreign-born are also shown, this reversal of the previous trend of decline is attributable to the steady increase of American-born Chinese. Even during the period when the total number of Chinese was decreasing, the growth of the American-born was persistent. In 1870, some 20 years after the first sizable influx of immigrants, there were hardly more than 500 American-born Chinese, amounting to less than 1% of the total Chinese population. Three decades later in 1900 the number of American-born, having increased nearly 20 times, totaled 9,010 and constituted 10% of the Chinese population.

In 1940, the American-born group outnumbered the foreign-born for the first time in the history of the Chinese population in the United States. (There is some evidence indicating biased reporting of nativity among foreign-born Chinese. So the census figures concerning American-born Chinese must be accepted with reservation.) In spite of the post-World War II immigration, the American-born continued to outrank the foreign-born in numerical prominence and comprised 53% of the total Chinese population in 1950.

Changes in sex and age composition

The great influx of Chinese occurred, as previously noted, between 1850 and 1890. Setting aside socio-economic factors which both stimulated and retarded Chinese immigration to this country (cf. Coolidge, '09; Sandmeyer, '39), this tremendous increase evidently placed the Chinese, identifiable easily in terms of racial and cultural traits, in the limelight and fostered the growth of an impressionalistic fear of "invasion by immigration." But, the Chinese were much less feared for what is termed "phenomenal fecundity" (Thomas, '52) than were the Japanese in this country.

Entering this country some 40 years later than the Chinese, the Japanese population had a large number of females by 1920. In that year, there were in the Japanese population some 24,000 foreign-born females out of a population of 111,000; whereas, the Chinese population, totalling 60,000, had only 2,500 foreign-born females. There were also some 14,000 and 5,000 American-born females in the Japanese and Chinese populations, respectively, who were then below the reproductive age. It was during this period that the charge of "phenomenal fecundity" was lodged against the Japanes (Strong, '34; Thomas, '52). Owing to a more tolerant attitude now, the Chinese probably will not be so stigmatized in the years to come, despite the growing number of females. Nevertheless, one of the reasons for which the Chinese were not feared for their "fecundity" in the 1920's and in the earlier years lay in the scarcity of females.

The number of females, as shown in table 1, has always lagged behind the number of males. In the 1860 census, a total of only 1,784 females was recorded; whereas, the number of males for that year was 33,149. In other words, there were 1,858.1 males per 100 females. From 1870 to 1910, the total number of females remained constantly under 5,000, while the number of males fluctuated violently. Thereafter, the males revealed a pattern of apparent stagnation, with a total of under 60,000 for three consecutive decades. During the same period from 1910 to 1940, the total number of females exhibited a trend of continuous growth, resulting in a more balanced sex distribution. The number of males per 100 females given in the census is as follows: 1,284.1, 1870; 2,106.8, 1880; 2,678, 1890; 1,887.2, 1900; 1,430, 1910; 695.5, 1920; 394.7, 1930; 285.3, 1940; and 189.6, 1950.

Foreign-born females were never more than a few thousand until 1950. American-born females, like American-born males, have increased steadily since the turn of the century. But, their numbers did not exceed 10,000 until 1930, and it was not until the end of the next decade that the total number of females reached 20,000, of whom 14,000 were American-born.

The scarcity of females, while reflecting the selectivity of migration in the decades prior to the adoption of the exclusion laws, was perpetuated by other immigration restrictions until recent years. Historically, therefore, the Chinese population was faced with the problem of replacement. As evident in the 1940 census, the total gain from 1930 amounted to only 2,600. Broken down by sex and nativity as shown in table 1, the over-all gain was the difference between the heavy loss of some 7,500 foreign-born males and the combined increase of 10,100 in the three remaining groups. Presumably, the mortality of the aging foreign-born male group, and possibly return migration to China, would more than likely have offset possible gains in the three other groups between 1940 and 1950.

Calculation of this sort, however, was upset by the relatively heavy immigration of Chinese after 1946. The gain of some 20,000 alien

Chinese not only served to bridge the gap of reproduction between the generations, but also helped to stabilize the age-sex structure of the Chinese population in the United States.

The consequences of the selectivity of, and the restrictions placed upon, the Chinese immigration in the nineteenth century are clearly shown in figure 1. The structure of 1900 consisted almost exclusively of males. Their age distribution, covering a wide range with a mode at 35–39 years of age, suggested a fairly old settlement. The age groups 15–19 and 20–24 were only a little larger than the age groups 60–64 and 65–69, reflecting the curtailment of immigration under the exclusion laws. The foundation of children was conspicuously small and, together with the barely visible female lateral, suggested the imbalance and instability of the population pyramid. The 1910 structure showed an enormous shrinkage of the male lateral from 1900. The female lateral remained about the same as a decade earlier. The pattern of instability persisted as the base of children was no larger than that of 1900.

The structures of 1920 and 1930 revealed the continuous contraction of the male lateral, notably in the age groups 45–49 and upwards. Certain other changes in the population pattern, however, also took place and were of significance. There appeared some expansion of the age groups 20–24 through 35–39 in both structures, reflecting the small amount of immigration in the two decades after 1910 (McEntire, '47). Evident also were the gradual growth of the female lateral and the enlargement of the base of children.

The development of a bimodal age distribution, for the males, was also suggested in the structures of 1920 and 1930. By 1940, the bimodal pattern of the males had sharpened, with the first mode at 15–19 years of age and the second mode at 40–44 years. For the females, owing to the selectivity of past immigration, a unimodal pattern persisted throughout the period up to and including 1940.

The 1940 structure again reflected the uninterrupted, but slow, expansion of the female lateral. Nevertheless, the foundation of children was smaller than in 1930, indicating a diminution in the rate of natural increase. While the aging of the first generation females and the immaturity of their American-born female descendants undoubtedly accounted for the decline, voluntary limitation of the family size was also responsible for the drop (Kowh, '47; McEntire, '47).

The structure of 1950 signified several far-reaching changes tending to balance and stabilize the Chinese population. While the bimodal pattern of the male persisted in 1950, their modes moved downward

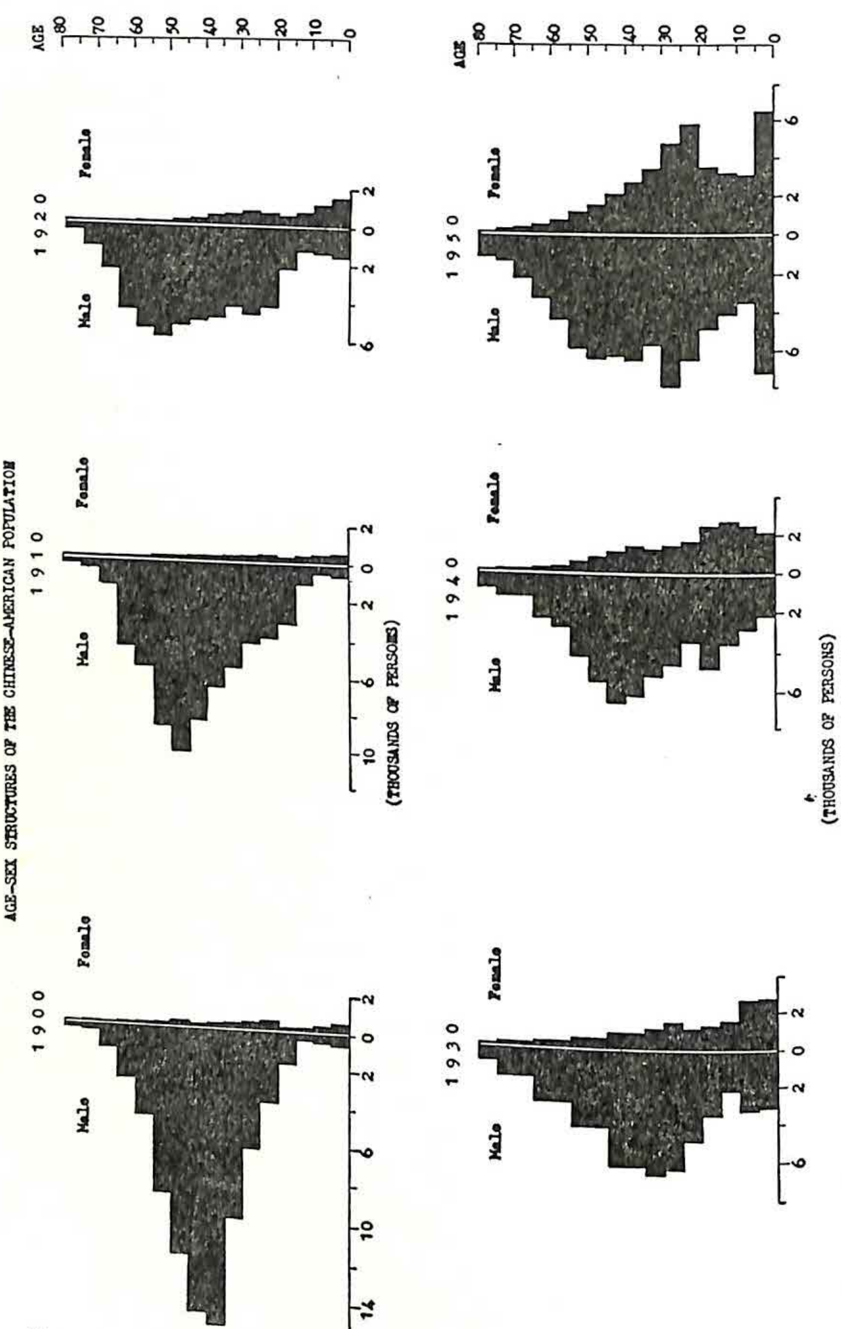
AGE-SEX STRUCTURES OF THE CHINESE-AMERICAN POPULATION

FIG. 1.

from 1940; the first mode descended from 15–19 years to 0–4 years, and the second from 40–44 to 25–29.

For the females, there developed in 1950 for the first time a bimodal pattern of distribution, with the first mode within the same age group as for the males and the second mode at the age group 20–24. The inflow of brides and other immigrants inflated the female lateral, notably in the age groups 20–24 through 45–49, and significantly equilibrated the population pyramid. The foundation of children, having expanded considerably, represented the contribution of these females and a high rate of natural increase.

TABLE 2

Births and deaths of the Chinese population, together with rates for the Japanese, 1940-1950 †

(*Rates per 1,000 estimated midyear population*)

	BIRTH			DEATH		
	CHINESE		JAPANESE	CHINESE		JAPANESE
YEAR	NUMBER	RATE	RATE	NUMBER	RATE	RATE
1950	5,139	43.9	24.5	*	*	*
1949	5,062	*	*	1,162	*	*
1948	4,210	48.4	23.2	1,162	13.4	6.7
1947	2,170	25.2	23.8	1,123	13.1	6.8
1946	1,534	18.5	21.5	1,279	15.8	7.0
1945	1,382	17.1	22.9	1,317	18.0	8.5
1944	1,291	16.3	22.9	1,324	17.9	7.6
1943	1,364	17.3	21.3	1,336	17.4	7.0
1942	1,220	15.6	17.3	1,206	15.7	7.4
1941	1,183	15.4	16.7	1,230	16.0	7.0
1940	1,098	14.1	14.7	1,184	15.2	6.7

Birth: For 1947-1950, based on population excluding armed forces overseas; for 1940-1946, on population including armed forces overseas.

Death: Exclusive of stillbirths and deaths among armed forces.

* Data not available.

† Source: *Vital Statistics—Special Reports*, U. S. Dept. of Health, Education and Welfare, PHS, National Office of Vital Statistics, Vols. 35, 36 and 37.

Table 2 gives the number of births and deaths of the Chinese from 1940 to 1950, inclusive, together with the rates for the Chinese and Japanese. These rates illustrate change in the two populations in recent years. After a decline in the rate of growth between 1930 and 1940, the Japanese resumed their upward trend in the 1940 decade, reflecting the coming of age of their American-born females. The 1950 census gave indication of only very small Japanese immigration since the end of World War II. The growth of the Japanese population in the 1940 decade therefore could not have been due to an increase of foreign-born females in the reproductive period.

As previously indicated, the Chinese population should have declined or remained stationary in the 1940 decade. Their birth and death rates between 1940 and 1945 unequivocally confirmed the correctness of the prediction. Beginning in 1946, however, the admission of Chinese females brought about the most startling change in the rate of natural growth: the birth rate for 1950 was nearly three times greater than the rate for 1940. The high birth rate of more than 40 per 1,000 is significant, particularly in view of the high sex-ratio of the Chinese which was 189.6 in 1950.

Although the crude death rate of the Chinese showed an apparent decline after 1945, it is very doubtful that the decline owed much to improved mortality conditions. While the number of deaths remained nearly the same during all the years of the 1940 decade, the decline in rate was obviously caused by the shifts of the population base upon which these rates were calculated.

In any event, from 1946 onward, the birth rate of the Chinese has far surpassed the death rate, and the number of births was far above replacement needs. With the majority of the foreign-born females still in the reproductive period and the coming of age of American-born females, the growth of the population in the next several decades can be certain if the mortality of the Chinese remains the same, or even shows some increase.

SUMMARY

The Chinese population was highly unstable and imbalanced at the beginning of the century. During the last 50 years, in spite of adverse demographic conditions, their numbers gradually increased. The rate of natural increase, however, did not contribute to the maintenance of the population size until after 1920. While the population grew in the two decades prior to 1940, it was believed that the Chinese population should have declined or remained stationary in the 1940 decade. The birth and death rates of the Chinese from 1940 to 1945 supported this view. But, the post-World War II immigration not only tended to balance the sex ratio of the Chinese, but also bridged the gap of reproduction between the generations. Consequently, a considerable increase of the Chinese was recorded in 1950. While it seems certain that the Chinese, with the passage of time, will exhibit the pattern of controlled fertility, there is no reason to assume that their numbers will not increase in the near future. The 1950 population of the Chinese contained the largest number of females and children ever known in the entire history of the population in this country. With the majority of foreign-born females still in the reproductive period and the coming of age of American-born females, a further growth in absolute numbers of the Chinese seems assured for at least several decades.

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VARIATIONS WITHIN INDIVIDUALS IN HUMAN BIOLOGY

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WHEN anatomical or physiological measurements are taken repeatedly on a single subject, the results obtained on different occasions will not usually agree exactly, even when conditions are carefully standardized. Discrepancies will result from actual errors of measurement, and these may be superimposed on real fluctuations in the quantity measured. In this article, both sources of variation will be referred to as *errors.* When real fluctuations are present, this terminology carries the implication that the subject possesses a *stable value,* about which random variations occur. For simplicity I leave aside the case in which the "stable" values actually change slowly with time; this can often be allowed for by a fairly simple extension of the analysis discussed here.

The assessment of errors

When some measuring technique is proposed, it is desirable to give some idea of its precision. Equally, in considering some physiological measurement it is of interest to know the extent to which this measurement fluctuates from time to time. Both questions can be included in the same investigation, but to simplify the discussion I shall consider only one aspect of the problem—either we are measuring a quantity which does not change with time (such as many anatomical measurements on adults) or we are investigating a variable quantity with instruments whose errors (at least with a single observer) can be ignored.

For an investigation of this kind we require that repeated measurements shall be taken by several observers on each of a number of subjects. It is important that the observers should form a representative sample of all possible observers, but the subjects may be chosen to give an even coverage of the whole range of measurements. In practice it is neither necessary nor desirable to have more than two measurements by each observer on each subject. It is assumed that the errors are equally variable at all points of the scale (a transformation of the scale of measurement may be necessary for this purpose); apart from statistical simplicity, this also makes the presentation of the results more straightforward. If necessary, the total number of measurements taken can be reduced by refinements in the experimental design, but this type of complication can easily be allowed for. In the simplest case, then, with p subjects and q observers, we can work out an analysis of variance as shown in table 1.

TABLE 1

	D. F	MEAN SQUARES	EXPECTED MEAN SQUARES
Subjects	$p-1$	V_S	
Observers	$q-1$	V_O	$2p\sigma^2_O+2\sigma^2_{SO}+\sigma^2_D$
Subjects × Observers	$(p-1)(q-1)$	V_{SO}	$2\sigma^2_{SO}+\sigma^2_D$
Duplicates	pq	V_D	σ^2_D

This in turn implies a model for the observations of the form

$$y=y_S+\epsilon_O+\epsilon_{SO}+\epsilon_D$$

y is the observed reading and y_S the stable value for the subject concerned, while the ϵ's relate to the particular observer, the particular observer-subject combination and the particular observation respectively. We assume further that the ϵ's are random variables, independently distributed with variances σ^2_O, σ^2_{SO} and σ^2_D. Then the expected values of the different mean squares are those shown in the last column of table 1.

The interpretation of the constants is quite straightforward. ϵ_D represents the error of measurement in the narrowest sense. ϵ_O, constant for a particular observer, allows for a constant personal bias. ϵ_{SO}, the interaction constant, is a little less familiar; it allows for the fact that an observer's bias may in practice vary from one subject to another. The assumption of independence is of some importance. It implies that the subject × observer interaction is not systematic—it is not the case, for example, that some observers always tend to over-estimate large measurements and under-estimate small measurements relative to others.

Estimates of the variance components are readily obtained from table 1. We have

$$s^2_D = V_D$$
$$s^2_{SO} = (V_{SO} - V_D)/2$$
$$s^2_O = (V_O - V_{SO})/2p.$$

If a single observer takes two measurements on the same subject, the difference between them is simply $\epsilon_{D1} - \epsilon_{D2}$; the standard deviation of such differences is therefore estimated by $\sqrt{2}s_D$. If two different observers are involved, however, the difference involves the ϵ_O's and ϵ_{SO}'s, and the standard deviation is estimated by $\sqrt{[2(s^2_O + s^2_{SO} + s^2_D)]}$.

Methods of reporting measuring errors

A variety of ways of reporting measuring errors can be found in the literature. Most authors have considered the case of a single observer. With duplicate measurements on p subjects we can simply form the p differences and calculate s^2_D as the (crude) sum of squares of the differences divided by p. This quantity is often referred to as s^2_{meas}. A more roundabout way of calculation that has been quite widely used is to find the correlation coefficient between the two sets of readings. Denoting the population value by ρ, several authors give the formula

$$\sigma^2_{\text{meas}} = \sigma^2(1 - \rho)$$

where σ^2 is the average variance of the two sets of readings. For this to be true, ρ must be the intra-class correlation (Fisher, '54), a qualification that is often overlooked. With duplicate readings, the much simpler difference technique is available; with more replicates, ρ is most easily calculated from the analysis of variance table, so that this method has little to recommend it.

ρ is commonly referred to as the "reliability coefficient" and there is some case for reporting a correct estimate of it when the accuracies of different measurements are being compared. For this purpose, it is essential to use a representative sample of subjects, since the estimate of σ^2 involves an estimate of the variance of the population of subjects. In general, however, estimates of the variance components are likely to be more useful.

The precision of variance components

It may be necessary to get some idea of the precision with which the various σ^2's have been estimated. The theory for calculating fiducial intervals for variance components was given by Fisher ('35), but the necessary tables have not been calculated. A usable approximation has been worked out by Bross ('50). Consider the estimate $s^2_O = (V_O - V_{SO})/2p$. We calculate the variance ratio $F = V_O/V_{SO}$ with $(q-1)$ and $(p-1)(q-1)$ degrees of freedom and obtain four values of F from tables (5% tables will give 90% fiducial limits). These are

F_O with $(q-1)$ and $(p-1)(q-1)$ d. f.
F'_O with $(q-1)$ and ∞ d. f.
F_1 with $(p-1)(q-1)$ and $(q-1)$ d. f.
F'_1 with $(p-1)(q-1)$ and ∞ d. f.

Then the lower limit is given by

$$(V_O - F_O V_{SO}) s^2_O / (F'_O V_O - F_O V_{SO})$$

and the upper limit by

$$(V_O - V_{SO}/F_1) s^2_O / (V_O/F'_1 - V_{SO}/F_1).$$

If the observed value of F is less than F_O the lower limit becomes negative and must be replaced by zero. In this case Tukey ('51) gives a better approximation to the upper limit as

$$(V_O - V_{SO}/F_1) s^2_O / (V_O/F'_1 - V_{SO}/F'_1)$$

which is most easily calculated as

$$(V_O - V_{SO}/F_1) F'_1/2p.$$

The effects of variation within subjects on regression and correlation coefficient

We now pass to the case where two different measurements are taken on each subject and interest centers on the relations between the measurements rather than on the variability as such. For this type of investigation it is necessary that the subjects should form a properly constituted sample, in which case we can define the variance of their stable values, σ^2_S, and estimate it from table 1 as $(V_S - V_{SO})/2q$. For simplicity, we consider the case of a single observer and duplicate readings on each measurement.

We now can construct an analysis of covariance as shown in table 2, denoting the two measurements by x and y

TABLE 2

		Mean Squares and Products		
	d. f.	x^2	xy	y^2
Subjects	$p-1$	A	C	E
Duplicates	p	B	D	F

We can estimate the variances of the stable values of x and y by $V_{xx}=\frac{1}{2}(A-B)$ and $V_{yy}=\frac{1}{2}(E-F)$ respectively, and in the same way their covariance by $V_{xy}=\frac{1}{2}(C-D)$. Then the regression coefficient of y on x (stable values) is estimated by V_{xy}/V_{xx}, that of x on y by V_{xy}/V_{yy} and the correlation coefficient by $V_{xy}/\sqrt{(V_{xx}V_{yy})}$. The effect of the errors on these coefficients should be noted. The estimate of the regression coefficient of y on x, calculated from a single series of observed values would be $(C+D)/(A+B)$. Now the errors will often be almost uncorrelated, so that D will be small, whereas B, being a variance, is essentially positive; consequently the regression coefficient is usually reduced in magnitude by the presence of errors in the independent variate. This is true of both regression coefficients and consequently of the correlation coefficient also.

This reduction in the size of the coefficients is fairly well known under the name of "attenuation," and various correction formulae, assuming uncorrelated errors, have been given. Yates ('53), for example, gives the corrected regression coefficient as the crude coefficient divided by $(1-h)$ where h is the ratio of the error variance to the total variance in the independent variate. Spearman's formula gives the corrected correlation as the crude coefficient divided by the product of the reliability coefficients. Both these formulae differ from our result only in terms of order $1/p$ that are quite negligible with the number of subjects usually encountered.

The precision of regression coefficients

When a regression coefficient has been calculated in the above way, some idea of its precision will often be needed. This and allied topics have been discussed in an important paper by Tukey ('51).

We may start with a slightly different problem. If the estimated correlation coefficient between the stable values is close to unity, it may be suggested that the stable values are *exactly* linearly related, the observed scatter being entirely done to the errors. Even when such a suggestion is implausible biologically, it may serve as a useful means of summarizing a set of results. The problem of determining the line of best fit has been discussed by statisticians and biologists, notably in connection with the allometric relationship; Tukey's approach appears to give a very satisfactory solution to a form of the problem that often arises in practice.

Suppose then that the true values lie on a line whose equation is $y-\mu x=c$; then any discrepancies between the observed values of $y-\mu x$ for the different subjects must be due to error. Now we do not know the value of μ; but we can replace it by a symbol m and work out an analysis of variance of $(y-mx)$ in the usual way. Values of m which give a non-significant variance ratio at a predetermined level of probability then define a fiducial range for μ; if no such real values exist, the data significantly contradict the hypothesis of strict linearity at the chosen probability level. When a point estimate of μ is required, this approach makes it natural to take the value of m which makes the variance ratio a minimum. This value is one root of a quadratic equation which is most easily written in the form of a determinant

$$\begin{vmatrix} 1 & m & m^2 \\ A & C & E \\ B & D & F \end{vmatrix} = 0.$$

The fiducial limits are the roots (if real) of the equation

$$\frac{\mu^2 A - 2\mu C + E}{\mu^2 B - 2\mu D + F} = \Phi$$

where Φ is the significance point of the F-distribution with $p-1$ and p d.f.

Returning to the estimation of the regression of the stable values of y on x, only approximate results are available. Tukey ('51) gives a formula for the variance of the regression coefficient, but this is not altogether satisfactory—for one thing, it does not give the correct answer in the absence of errors—and I suggest as an alternative

$$\mathrm{Var}(b) = \frac{\dfrac{A}{p-1}\left(E - \dfrac{(C-D)^2}{A-B}\right) + \dfrac{BF}{p}}{(A-B)^2}.$$

Numerical example

As an example of some of the methods discussed above, I use readings of oral and rectal temperatures taken on two occasions about a week apart on 46 young men (Tanner, '51). The "errors" here are mostly physiological fluctuations, the errors of measurement being comparatively small. The analysis of covariance gives the mean square and products shown in table 3.

TABLE 3

	d. f.	ORAL (°F) x^2	xy	RECTAL (°F) y^2
Subjects	45	0.31119	+0.25443	0.29209
Duplicates	46	0.04685	+0.03424	0.07489
Stable values		0.1322	+0.1101	0.1086

Considering first the variates separately, the "errors" in oral temperatures are distributed with a standard deviation estimated by $\surd(0.04685) = 0.216°$. This means that two readings of oral temperature taken on the same subject at a weekly interval should not differ by more than $1.96 \times \surd 2 \times 0.216 = 0.60°$ in 95% of all cases. Put another way, a single reading of oral temperature can be expected to fall within $\pm 0.42°$ of the stable value 95% of the time.

The stable values are distributed with variances and covariance whose estimates are given in the last line of table 3—here, for example, $0.1322 = \frac{1}{2}(0.31119 - 0.04685)$. The mean of all the oral temperatures was 98.08° so that we expect 95% of the population to have stable oral temperatures in the range $98.08 \pm 1.96 \surd(0.1322) = 97.37°$ to 98.79°. The variance of a set of single readings is estimated as $0.1322 + 0.0468 = 0.1790$, giving a standard deviation of 0.42°.

To set limits on the variances of the stable values, we find from tables of $2\frac{1}{2}\%$ significance points

$$F_0 = 1.80 = F_1$$

$$F_1 = 1.45 = F_1$$

so that for oral temperatures we have the lower limit

$$\frac{0.31119 - 0.04685 \times 1.80}{0.31119 \times 1.45 - 0.04685 \times 1.80} \times 0.1322 = 0.0817$$

and the upper limit

$$\frac{0.31119-0.04685/1.80}{0.31119/1.45-0.04685/1.80}\times 0.1322=0.1998$$

Tukey's modification gives an upper limit of 0.2067.

Turning to the relationship between the two variates we have for the stable values

$$\begin{aligned} &\text{regression coefficient of } y \text{ on } x = +0.1101/0.1322 = +0.833 \\ &\text{regression coefficient of } x \text{ on } y = +0.1101/0.1086 = +1.014 \\ &\text{correlation coefficient} \qquad = +0.1101/\sqrt{(0.1322\times 0.1086)} \\ &\qquad\qquad\qquad\qquad\quad = +0.919. \end{aligned}$$

These may be compared with the "attenuated" values that would have been obtained from a series of single measurements, $+0.806$, $+0.786$ and $+0.796$ respectively.

If we wish to estimate a strict linear relationship of the form $y-\mu x=c$, we estimate μ from the equation

$$\begin{vmatrix} 1 & m & m^2 \\ 0.31119 & 0.25443 & 0.29209 \\ 0.04685 & 0.03424 & 0.07489 \end{vmatrix}=0.$$

This gives $0.009053-0.009621m-0.001265m^2=0$, and the appropriate root is $m=+0.8466$. With this value, the analysis of variance for $y-mx$ is

	d. f.	*MS*	*F*
Subjects	45	0.08433	1.670
Duplicates	46	0.05049	

The variance ratio lie just beyond the 5% level of significance. For illustrative purposes, I calculate the 1% fiducial limits. These are the solutions of

$$\frac{0.31119m^2-0.50886m+0.29209}{0.04685m^2-0.06848m+0.07489}=2.01$$

or

$$0.21702m^2-0.37122m+0.14156=0$$

giving $m=0.574$ to 1.137.

Returning to the regression of y on x, the estimated coefficient is 0.833. The variance of this quantity is approximately estimated as

$$\frac{\frac{0.31119}{45}\left(0.29209-\frac{0.22019^2}{0.26434}\right)+\frac{0.04685\times 0.07489}{46}}{(0.26434)^2}=0.01185,$$

giving a standard error of ± 0.109.

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CONTRIBUTION TO THE STUDY OF THE QUESTION OF ASSOCIATION BETWEEN TWO DISEASES *

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1. INTRODUCTION

THIS paper deals with some questions on the association between two diseases: how to define this concept and how to assess it. These considerations found their origin in the Research Seminar on Statistical Problems of Health, directed by Dr. J. Neyman, held at the Department of Statistics, University of California, Berkeley, during the summer of 1956. The subject of this paper was inspired by the Pearl-Wilson problem of the association between cancer and tuberculosis, Pearl ('29), Wilson ('30), Wilson and Maher ('32). However, the discussion applies equally well to any pair of diseases, so that for the sake of generality we shall denote these two diseases by A and B. The main part of the paper is concerned with a discussion of various possible definitions of association, their inter-relation, and their evaluation. In the Appendix a method is presented for dealing mathematically with the relevant stochastic models.

2. DEFINITIONS OF ASSOCIATION BASED ON FUNDAMENTAL PROBABILITY SETS

There is more than one way in which the concepts independence, positive association and negative association between diseases A and B can be defined, and it is therefore quite important to be clear about which definition one should adopt. First of all one can focus attention

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on a certain well-defined group of individuals and sort this group into 4 categories, denoted, respectively, by O, A, B and AB: those individuals who have neither A nor B, those who have A only, those who have B only, and those who have both. Let the numbers of indiivduals in these categories be n_O, n_A, n_B and n_{AB} and define the ratio R_1 as

$$(1) \qquad R_1 = \frac{n_O n_{AB}}{n_A n_B} .$$

Any such group of individuals on which the ratio R_1 is based is sometimes called a "Universe of Discourse" Wilson ('30), sometimes a "Fundamental Probability Set," Neyman ('50), abbreviated FPS. We shall adopt the latter nomenclature. In whatever FPS we have adopted, the diseases A and B are said to be positively associated if $R_1 > 1$, independent if $R_1 = 1$ and negatively associated if $R_1 < 1$. An equivalent definition can be give nin terms of the ratio

$$R'_1 = \frac{n n_{AB}}{n'_A n'_B}$$

in which $n = n_O + n_A + n_B + n_{AB}$ is the total number of individuals in the FPS, $n'_A = n_A + n_{AB}$ is the number of individuals having disease A, and $n_B = n'_B + n_{AB}$ is the number having B. The two ratios R_1 and R'_1 are either both > 1, or both $= 1$ or both < 1, so that the definition of association using R'_1 coincides with the definition using R_1.

Examples of the above definition may be found in the papers by Pearl ('29), Wilson ('30) and Wilson and Maher ('32). The FPS considered by Pearl consisted of all individuals who had died in a certain hospital during a certain period, and the classification into the 4 categories was done by determining at autopsy the presence or absence of A or B. Pearl found that with respect to this particular FPS cancer and active lung T.B. are negatively associated and suspected that this result has various practical implications. These findings were criticized by Wilson who showed that one can define more than one FPS, compute R_1 (or R'_1) for each of them and arrive at contradictory conclusions. Thus, he first took the FPS composed of all individuals who had died in the United States during a certain year and sorted them into the 4 categories. In this population the ratio R_1 showed that A and B were negatively associated, as in Pearl's case. Next Wilson took the FPS of all individuals who were alive at some time during the year under consideration. Those who died by the end of the year were sorted as before; those who were still alive (*i.e.*, the great majority) were classified as having neither A nor B. In this FPS the ratio R_1 showed positive association. Since the two procedures lead to opposite conclusions, Wilson puts the question as to which one is correct.

At this point it may be remarked that one may have some objection to Wilson's second procedure for the following reason. Whatever definition one wants to adopt for the association between diseases A and B, it should not depend on the period of observation T. In Wilson's examples this period was $T = 1$ year, but it could equally well have been, say, half year or two years. Now in equation (1) the quantities n_A, n_B and n_{AB} represent the numbers of individuals in the various categories, having died during the period T, so that these quantities are approximately proportional to T. On the other hand, in Wilson's second example n_O is practically equal to the size of the live population, and is therefore approximately independent of T. Hence, by this procedure R_1 would be approximately inversely proportional to T. In other words, observations over a short period would make one believe that A and B are positively associated, whereas observations over a long period would lead to the conclusion that A and B are negatively associated. Obviously, this is not a satisfactory procedure. However, Wilson himself considers still further procedures to define association, all of them considering the individuals alive with either disease. The procedure most favored by Wilson is to abandon basing the definition of association on mortality data altogether, and instead, base it on morbidity data alone. This means that the FPS to be adopted should consist solely of the individuals alive at a certain time and should be sorted into the 4 categories O, A, B, AB. Association is then defined, as usual, in terms of the ratio R_1, computed from (1). It will be seen in section 4 that the ratio R_1, so computed, is the empirical determination of the ratio R_2, given by (8), or of its probabilistic analogues given by the left-hand sides of (12) and (14). For a discussion of the value of these ratios we refer to section 5.

3. DEFINITIONS OF ASSOCIATION BASED ON MATHEMATICAL MODELS

It cannot be denied that the evaluation of various percentages relating to certain classes of individuals in the general population does present some interest. In particular, Pearl's finding of a negative association between cancer and tuberculosis among the deceased is certainly interesting. However, similar studies acquire a particular interest and, occasionally, practical importance if they are not performed for their own sake but as a means for finding something about the mechanism behind a phenomenon. In the particular case of discussion between Pearl and Wilson, the relevant phenomenon that both authors had undoubtedly in mind is the phenomenon of acquiring either cancer or tuberculosis, or both, and eventually succumbing to them. Thus, one of the most relevant questions is whether the contracting of a disease A by an originally healthy person increases, decreases, or leaves unchanged his chances of contracting also the disease B. The same question can be asked with A and B interchanged. Finally, the question arises whether the risk of death of a person having both A and B is different from what would be expected on the basis of the death risks from A and B separately. The following pages are written with these particular questions in mind. It will be noticed that these questions are related to the contrasting concepts of prevalence and incidence of diseases that are the subjects of current discussions by a number of authors, such as Dorn ('51), Sartwell and Merrell ('52) and Neyman ('55).

$$\begin{tikzcd}
S_1 & & & S_3 \\
& S_0 \arrow[ul] \arrow[r] \arrow[d] & S_2 \arrow[ur] \arrow[d] & \\
& S_4 \arrow[dl] \arrow[r] & S_6 \arrow[dr] & \\
S_5 & & & S_7
\end{tikzcd}$$

FIG. 1. DIAGRAM UNDERLYING THE SIMPLEST MATHEMATICAL MODEL TO REPRESENT THE PHENOMENON OF ACQUIRING DISEASES A AND B AND SUCCUMBING TO THEM.

The method of approach to the problem of association between the diseases A and B, as presented in this and the following sections, requires the construction of a mathematical model of the phenomenon of acquiring the diseases in question and of succumbing to them. An example of such a model can be found in the treatment of recovery, relapse, death and loss of patients by Fix and Neyman ('51). The model represented by the diagram in figure 1, proposed by Dr. J. Neyman, is one of the simplest models relevant to the question of association between diseases A and B. We postulate that at any time (t) each individual of the population under consideration must be in one of the following exclusive states:

S_0 = state of being alive without A and without B.

S_2 = state of being alive with A but without B.

$S_4 =$ state of being alive without A but with B.
$S_6 =$ state of being alive with both A and B.
$S_1 =$ state of being dead, having been in S_0 just prior to death.
$S_3 =$ state of being dead, having been in S_2 just prior to death.
$S_5 =$ state of being dead, having been in S_4 just prior to death.
$S_7 =$ state of being dead, having been in S_6 just prior to death.

As time goes on, an individual, originally in a particular state S_i, may transfer to another state S_j. Also, certain states (those of being dead) are "terminal," or "absorbing," and no transitions from them are possible. The detailed assumptions regarding the transitions from one state to another constitute the model. In relation to the problem of association between the two diseaeses A and B, the simplest assumptions regarding the transitions are indicated by arrows in figure 1. It will be seen that in this model no transition is reversible, *i. e.*, if an individual leaves a state S_i, then no return to this state is possible. This implies that each of the diseases A and B is so dangerous as to preclude recovery.

The diagram of figure 1 will first be used as the basis for a deterministic model. Instead of considering the number of individuals in the various states, we shall think of a substance present in and flowing between the various states. At a certain fixed time let the amount of substance in state S_i be x_i $(i = 0, 2, 4, 6)$ and let the amount flowing from S_i to S_j per unit time be proportional to x_i, with proportionality constant q_{ij}. The product $x_i q_{ij}$ represents then the flow per unit time from S_i to S_j. The model is completely determined by specifying the values of the flow intensities q_{ij}. It should be noted that several q_{ij} are equal to zero, namely, those not corresponding to an arrow in figure 1. For convenience of terminology we shall call $q_{02}, q_{04}, q_{26}, q_{46}$: *incidence intensities*, and $q_{01}, q_{23}, q_{45}, q_{67}$: *mortality intensities.*

It is possible now to define association between diseases A and B in terms of assertions on the values of the q_{ij}. In fact, there are two concepts which have to be distinguished: association between incidence of A and B, and association between mortality from A and B. This leads to three definitions, given below.

DEFINITION 1. We shall call A and B *incidence-independent* if

$$q_{02} = q_{46} \text{ and } q_{04} = q_{26}. \tag{2}$$

Positive and negative incidence association can be defined by replacing the equalities in (2) by the appropriate inequalities. The interpretation of (2) is that if A and B are incidence-independent, then the risk of acquiring B is independent of whether or not A is present; and the same statement with A and B interchanged.

Before defining mortality-independence we shall make the assumption that an individual possessing a certain disease has, in addition to the risk of dying from this disease, independently a risk of dying from other causes, and we assume, moreover, that the latter risk is equal to that of a healthy person. These assumptions are expressed in the following analysis of q_{23}, q_{45} and q_{67}

$$\begin{aligned} q_{23} &= q_{01} + q'_{23} \\ q_{45} &= q_{01} + q'_{45} \\ q_{67} &= q_{01} + q'_{67} \end{aligned} \tag{3}$$

in which q'_{23}, q'_{45} and q'_{67} correspond to the risk of dying from A alone, B alone, or AB respectively.

DEFINITION 2. We shall call A *and* B *mortality-independent* if

$$q'_{67} = q'_{23} + q'_{45} \tag{4}$$

expressing the additivity of the risk intensities of the two independent causes of death.

DEFINITION 3. A and B will be called *completely independent* if they are both incidence- and mortality-independent.

The diagram in figure 1 will now be used for a stochastic model. At time $t = 0$ (birth) an individual starts in state S_0 and in the course of time he may make transitions to other states along the arrows indicated in figure 1. The time t is identified with the age of the individual. The transition intensities will be denoted again by q_{ij}.[1] We shall assume the same analysis, given by (3), of q_{23}, q_{45} and q_{67}. Unlike in our deterministic model, the q_{ij} will in general not be constants, but depend on the age t. In addition q'_{23} and q_{26} may also depend on how long the person has had disease A. Similar remarks apply to q'_{45}, q_{46} and q'_{67}. In other words, some of the q's may be both age dependent and duration dependent (the duration referring to the disease or diseases). Mathematically this is conveniently handled as follows.

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[1] The exact meaning of the transition intensity q_{ij} is as follows: For a small interval of time Δt, the conditional probability that an individual in state S_i at time t will be transferred into S_j before the moment $t + \Delta t$, is given by $q_{ij}\Delta t + o(\Delta t)$.

If disease A is contracted, let u be the time at which this happens. Similarly, if B is contracted, let v be the time at which this happens. It is, of course, possible that both events happen in succession, which has the result that the individual enters S_6. Then, in general, q'_{23} and q_{26} are functions of t and u, q'_{45} and q_{46} of t and v, and q'_{67} of t, u and v. On the other hand, q_{01}, q_{02} and q_{04} will be only functions of t. Incidence- and mortality-independence will be defined by equations analogous to (2) and (4). Thus, we have as definition of incidence-independence

$$q_{02}(t) = q_{46}(t, v), \qquad q_{04}(t) = q_{26}(t, u) \tag{2'}$$

identically in t, u, v, and as definition of mortality-independence

$$q'_{67}(t, u, v) = q'_{23}(t, u) + q'_{45}(t, v) \tag{4'}$$

identically in t, u, v.

As was said before, the model based on the diagram of figure 1 does not allow recovery from either A or B. A more general model is based on the diagram presented in figure 2. The diagram is understood to extend indefinitely to the right. The meaning of the states $S_1, \cdots, S_7$ is as before. $S_2{}^{(1)}$ can be taken either as a state of being recovered from disease A, or as a state of having A in an inactive form (as in tuberculosis). $S_2{}^{(2)}$ is a state of relapse into the active form of A, etc. The states S_6, $S_6{}^{(1)}$, etc. have the same meaning as the corresponding states S_2, $S_2{}^{(1)}$, etc., with the additional specification that disease B is also present. The states labeled with an odd subscript denote again the corresponding state of being dead. The model could be extended to allow for a similar recovery-relapse sequence in patients with disease B, but this will not be done here. The diagram in figure 2 can be used either for a deterministic or for a stochastic model. In either case,

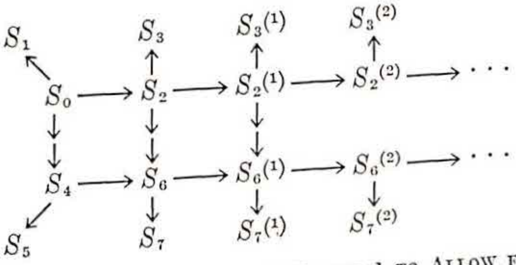

FIG. 2. EXTENSION OF THE DIAGRAM IN FIGURE 1 TO ALLOW FOR A RECOVERY-RELAPSE SEQUENCE IN DISEASE A.

the association between A and B can be defined in terms of the flow intensities or the transition intensities, as the case may be, between the states. Specifically, incidence-independence is defined as equality of the intensities $S_0 \rightarrow S_4$, $S_2 \rightarrow S_6$, $S_2^{(1)} \rightarrow S_6^{(1)}$, etc., equality between $S_0 \rightarrow S_2$ and $S_4 \rightarrow S_6$, equality between $S_2 \rightarrow S_2^{(1)}$ and $S_6 \rightarrow S_6^{(1)}$, etc. Mortality-independence is defined with the help of (4) or (4′) and similar relations.

4. SOME CONSEQUENCES OF THE MATHEMATICAL MODELS

Of the deterministic model based on figure 1, section 3, we shall seek a steady state solution, *i. e.*, a solution based on the assumption that the x_i are constant in time. This would be a representation of a population in dynamic equilibrium. In order to keep the system in a steady state, the hypothetical substance substituted for the population has to be supplied into state S_0 at a constant rate, say b. In a human population b would be the birth and immigration rate. For convenience of notation, for each S_i we define Q_i by

$$Q_i = \sum_j q_{ij}. \tag{5}$$

In a steady state the flow into any state S_i equals the flow out of that state. This leads to the following system of 4 equations:

$$\begin{aligned} &x_0 Q_0 = b \qquad && x_4 Q_4 = x_0 q_{04} \\ &x_2 Q_2 = x_0 q_{02} && x_6 Q_6 = x_2 q_{26} + x_4 q_{46}. \end{aligned} \tag{6}$$

These 4 equations determine the "state frequencies" x_i if the transition intensities q_{ij} and the birth and immigration rate b are given. In fact, to each set of positive b and q's there corresponds a unique set of positive x's. However, the q_{ij} are unobservable whereas, in principle, the x_i can be measured. It is therefore more realistic to consider the x_i as known, and (6) as a system of equations in the unknown q_{ij}. Unfortunately, there are fewer equations than quantities q_{ij}, so that some of the latter have to be considered as known in (6). Since there are 4 equations (6), it would seem as if we could arbitrarily select 4 of the q_{ij} to be the unknowns. However, if we select the 4 incidence intensities, then only 3 of the 4 equations (6) turn out to be independent. On the other hand, if we take three incidence intensities and one mortality intensity to be the unknowns, then the 4 equations (6) are independent. For example, with the help of (6) it is possible to compute, say, q_{04}, q_{26}, q_{46} and q_{67} in terms of the remaining q's and the x's, with the result:

$$\begin{aligned} q_{04} &= x_0^{-1}[b - x_0(q_{01} + q_{02})] \\ q_{26} &= x_2^{-1}[x_0 q_{02} - x_2 q_{23}] \\ q_{46} &= x_4^{-1}[b - x_0(q_{01} + q_{02}) - x_4 q_{45}] \\ q_{67} &= x_6^{-1}[b - x_0 q_{01} - x_2 q_{23} - x_4 q_{45}]. \end{aligned} \tag{7}$$

It follows that in order to answer all questions regarding the transition intensities, it is necessary to estimate empirically at least three mortality intensities (*e.g.*, q_{01}, q_{23}, q_{45}) and one incidence intensity (*e.g.*, q_{02}), in addition to the "state frequencies" x_0, x_2, x_4, x_6 and the birth and immigration rate b. The remaining transition intensities can then be computed and questions regarding incidence- and mortality-dependence answered.

It should be remarked that mathematically it is immaterial which incidence intensity and which set of three mortality intensities are chosen to be the known quantities. The choice should therefore depend on practical considerations, and the choice we made above, leading to equations (7), may in a particular situation not be the most practical one. For instance, if q_{67} can be estimated more easily than q_{01}, the latter should appear on the left-hand side of the last equation (7) and the former on the right-hand side of equations (7). Similarly, if, say, q_{46} can be estimated more easily than q_{02}, the third equation (7) should be replaced by a similar equation for q_{02}, whereas q_{46} would appear on the right-hand side of equations (7).

Corresponding to the ratio R_1, given by (1), there are two ratios in our present mathematical model which are of interest:

$$R_2 = \frac{x_0 x_6}{x_2 x_4} \tag{8}$$

and

$$R_3 = \frac{(x_0 q_{01})(x_6 q_{67})}{(x_2 q_{23})(x_4 q_{45})} = \frac{q_{01} q_{67}}{q_{23} q_{45}} R_2. \tag{9}$$

With reference to the discussion in section 2, the ratio R_2 would be taken as a measure of association between the diseases A and B in the FPS composed of all those members of the population that are alive. On the other hand, R_3 represents a similar measure referring to the population of those that died during a specified period of time. With the help of (6), both ratios can be expressed in terms of the transition intensities. For R_2 we get

$$R_2 = \frac{1}{q_{67}} \left(\frac{q_{46}}{q_{02}} Q_2 + \frac{q_{26}}{q_{04}} Q_4 \right) \tag{10}$$

with the Q_i given by (5). R_3 follows after substitution of (10) into (9).

One of the important questions is whether either R_2 or R_3 enables us to decide whether or not the diseases A and B are independent. This question will be taken up more fully in section 5. Here we restrict ourselves to consider what happens if A and B are completely independent (see definition 3). Using (2) and (4) in (10) we obtain

$$R_2 = 1 + Q_0/q_{67} \tag{11}$$

in the case of complete independence. Considering that Q_0 is the relative rate at which healthy persons die or get sick with A or B, whereas q_{67} is the relative death rate of persons sick with A and B, we may expect that in an actual situation Q_0/q_{67} is small compared to unity. If that is the case, then it follows from (11) that R_2 is only slightly greater than unity. That R_2 does not equal unity exactly is a consequence of the fact that in our deterministic treatment we consider in each state a mixture of all age groups. In the probabilistic treatment, which follows next, the fate of a single age group is studied, and it will be seen that the ratio analogous to R_2 has a value exactly equal to unity if A and B are completely independent.

Turning now to the ratio R_3, we see from (9) that the factor by which we have to multiply R_2, in order to obtain R_3, equals $q_{01}q_{67}/(q_{23}q_{45})$. Since q_{01} is the relative death rate of healthy persons, whereas the remaining q's are very nearly the relative death rates of sick persons, we may expect the above ratio to be small compared to unity. As a result, we may expect R_3 to be much less than unity, even in the case of complete independence.

Next we shall treat the stochastic model, based on figure 1 or on figure 2. Let an individual start in state S_0 at time $t = 0$, and let $P_i(t)$ denote the probability of being in state S_i at time t $(i = 0, \cdots, 7)$. In the model of figure 2 we also have the probabilities of being in the states $S_2^{(1)}, S_2^{(2)}, \cdots$, and $S_6^{(1)}, S_6^{(2)}, \cdots$, at time t. Let these probabilities be denoted by $P_2^{(1)}(t), P_2^{(2)}(t), \cdots$, and $P_6^{(1)}(t), P_6^{(2)}(t), \cdots$. In the Appendix a mathematical treatment of the stochastic models will be given, and it will be shown that the following holds if A and B are completely independent:

$$\frac{P_0(t)P_6(t)}{P_2(t)P_4(t)} = 1 \tag{12}$$

The left-hand side of (12) is the probabilistic analogue of the deterministic ratio R_2, defined by (8). We see then that in the stochastic model the ratio of interest is exactly equal to unity in the case of complete independence. In the model based on figure 2 we obtain for any m

$$\frac{P_0(t)P_6^{(m)}(t)}{P_2^{(m)}(t)P_4(t)} = 1. \tag{13}$$

Whereas $P_2(t)$ is the probability of having the disease A at time t for the first time, with B being absent, $P_2^{(2)}(t)$ is the probability of having A at time t for the second time, with B absent, etc. One would probably be most interested in the probability of having A and not B at time t, without qualification on the number of intermediate relapses. Let this probability be denoted by $P'_2(t)$, then

$$P'_2(t) = P_2(t) + P_2^{(2)}(t) + P_2^{(4)}(t) + \cdots.$$

Similarly, let $P'_6(t)$ be the probability of having A and B at time t, without qualification on the number of intermediate relapses. Then from (13) we obtain easily

$$\frac{P_0(t)P'_6(t)}{P'_2(t)P_4(t)} = 1 \tag{14}$$

and again we get the interesting ratio exactly equal to unity.

The probabilistic analogue of the ratio R_3, defined by (9), has the same relation to the ratio on the left-hand side of (12) (or of (14) as the case may be) as R_3 has to R_2, so that the same conclusions hold for this ratio as for R_3.

5. DISCUSSION AND CONCLUSIONS

We return to the question whether the presence or absence of dependence between the diseases A and B can be decided by the value of a ratio like R_2 or R_3, given by (8) and (9), or their probabilistic analogues. First of all it is clear that R_3 is useless, since even in the case of complete independence it is not even close to unity. The same remark applies, of course, to the probabilistic analogue of R_3. Remembering that R_3 is the ratio R_1 given by (1), based on an FPS composed of individuals who have died during a certain time interval, it is possible then to observe a value for this ratio which is much less than unity. One is tempted then to conclude negative association between A and B, (Pearl, '29), whereas in reality A and B may be completely independent.

The following numerical example will illustrate this. It should be understood that the numbers chosen are purely fictitious. Let in a particular year in a certain country (say the United States) the number of individuals of age 30 years (reported age) in the states S_0, S_2, S_4 and S_6 be 1,000,000; 10,000; 10,000 and 100 respectively. The ratio $1{,}000{,}000 \times 100/(10{,}000 \times 10{,}000) = 1$ is in accordance with (12) and is therefore fully compatible with complete independence of A and B. Suppose that indeed A and B are completely independent, and that $q_{01} = .0001$, $q'_{23} = .02$, $q'_{45} = .03$, so that, by (3) and (4), $q_{23} = .0201$, $q_{45} = .0301$ and $q_{67} = .0501$. The expected number of individuals of age 30 years dying during the year of observation in each of the 4 categories is $1{,}000{,}000 \times .0001 = 100$; $10{,}000 \times .0201 = 201$; $10{,}000 \times .0301 = 301$ and $100 \times .0501 = 5$. The cross ratio of these numbers is

$$100 \times 5/(201 \times 301) = .00826$$

which is much less than unity. If we would have based the definition of association on this ratio, we would have concluded strong negative association in flagrant disagreement with our assumption of complete independence in terms of the mathematical model.

Next, let us see how useful the ratio R_2 is, or its probabilistic analogues given by the left-hand sides of (12) and (14). For simplicity of discussion we shall in the following denote by R_2 either of the expressions given by (8) and the left-hand sides of (12) and (14), and we shall pretend that the right-hand side of (11) equals unity exactly. We have shown then in section 4 that complete independence implies $R_2 = 1$, so that at first glance it seems as if this ratio is a meaningful measure of independence. At closer inspection, though, it becomes clear that as a measure of independence R_2 cannot be used. The reason for this is that although complete independence implies $R_2 = 1$, the converse is by no means true. For instance, if we take in (10): $q_{02} = q_{04} = .0001$, $q_{26} = q_{46} = .0002$, $q_{23} = q_{45} = .02$, $q_{67} = .08$ (arbitrary units), then we compute $R_2 = 1.01$, whereas neither (2) nor (4) are fulfilled. As a matter of fact, the particular choice of q's implies that the incidence intensity of, and mortality intensity from disease A, is twice as high in the presence of B as it is in its absence and similarly with A and B interchanged. Thus, there is strong incidence dependence as well as mortality dependence, both in the same direction, but yet the ratio R_2 is very close to unity. Incidentally, in the above example we chose $q_{46}/q_{02} = q_{26}/q_{04}$, which means in words that the presence of B increases the incidence rate of A by the same factor as the presence of A increases the incidence rate of B. There is little justification for this choice, though, and we may even conceive of the possibility that $q_{46} > q_{02}$ and at the same time $q_{26} < q_{04}$, which would mean that the incidence rate of A is increased by the presence of B (over what it is in the absence of B), whereas the incidence rate of B is decreased by the presence of A. In that case the incidence-dependence of A and B could not any longer be described as either positive or negative.

From the preceding paragraph it would seem that the ratio R_2 is as useless as the ratio R_3 for the problem of investigating possible dependence between the diseases A and B. For, if R_2 is determined empirically, and found to be close to unity, then no conclusion can be drawn at all. However, if R_2 is found to be very different from unity,, then we are able to draw a definite conclusion, namely that the diseases A and B are not independent. The situation is the same here as in many testing problems in statistics, where the power of the test is either unknown, or known to be small for certain classes of alternatives. If the observations lead to acceptance of the hypothesis, we will tend to consider this inconclusive rather than believe that the hypothesis is true. On the other hand, if the observations lead to rejection of the hypothesis, then we do conclude that the hypothesis is false, our confidence in the correctness of this conclusion being inversely proportional to the error probability associated with the hypothesis. In our case, the hypothesis would be that A and B are completely independent, and if the test would be based on R_2 alone, the acceptance region would presumably be an interval about unity. For certain alternatives, such as the one given in the numerical example, the probability of accepting a false hypothesis would be very high.

From the foregoing discussion it follows that it is not completely meaningless to look at the ratio R_2, for if it turns out to be very different from unity we can make a definite decision. However, even if this happens, we can only infer that A and B are not completely independent, but we cannot tell in which way they are dependent. It follows then that in order to answer all questions concerning dependence, it is not sufficient to observe only R_2. With reference to section 4, specifically equations (7), it is necessary not only to estimate empirically x_0, x_2, x_4 and x_6, but also three mortality intensities and one incidence intensity, *e.g.*, q_{01}, q_{23}, q_{45} and q_{02}. Then and only then can all questions regarding dependence be settled. It should be remarked that this situation is bad, because it requires data of a kind not usually available.

The conclusions of the preceding paragraph were drawn under the presumption that the observations are made either on a population of mixed ages, or on one narrow age group. In either case one obtains fewer equations than unknown transition intensities q_{ij}. The situation may possibly improve considerably if observations are made on several age groups. With reference to the stochastic model based on figure 1, section 3, suppose that the assumptions concerning the dependence of the q_{ij} on t, u and v are reasonably simple, *i. e.*, involving some, but not too many parameters, and suppose that the $P_i(t)$ $(i=0,2,4,6)$ are computed in terms of the unknown parameters and t. Keeping in mind that mortality data are more easily available than morbidity data, we can equate the theoretical expressions for the expectations of the various death rates, such as $P_0(t)q_{01}(t)$, $P_2(t)q_{23}(t)$, etc., to the observed death rates for age t. If death rate data are available for enough age groups, then possibly enough equations are obtained to make possible the estimation of the unknown parameters in the functions q_{ij}. This needs further investigation.

APPENDIX

Mathematical treatment of the stochastic models

First, it will be indicated how an explicit solution can be obtained to a Markov process represented by a diagram which has no closed paths. The diagrams in figures 1 and 2 satisfy this requirement. The solution may be obtained at once in the form of certain integrals, without the necessity of writing down and solving differential equations. For simplicity the method will be illustrated for the process represented by the diagram in figure 1 and only in the case of complete independence. The treatment of the more general situations wil lthen be obvious.

The method starts by introducing certain conditional densities and certain functions which may be called "survival functions." Remembering that, in section 3, u was defined as the time at which the transition $S_0 \to S_2$ is made, if it does in fact occur, we introduce the conditional density $p_2(u \mid t)$ of u, given that at t the individual is in state S_2 (obviously $p_2=0$ unless $u \leqq t$). The densities $p_4(v \mid t)$ and $p_6(u, v \mid t)$ are defined in a similar way. The notation of section 3 will be changed slightly: q_{01}, q'_{23} and q'_{45} will be replaced by q_1, q_3 and q_5 respectively, $q_{02}=q_{46}$ by q_2 and $q_{04}=q_{26}$ by q_4. It should be remembered that all q_i depend on t: q_3 depends also on u and q_5 depends also on v. In order to simplify the writing we shall suppress in the notation the dependence of q_3 on u and of q_5 on v. The following functions are introduced:

$$L_i(t_1, t_2) = \exp[-\int_{t_2}^{t_2} q_i(x)\,dx] \qquad i = 1, \cdots, 5. \tag{15}$$

Obviously the functions L_i satsfy the following property:

$$L_i(t_1, t_2) L_i(t_2, t_3) = L_i(t_1, t_3) \quad \text{if} \quad t_1 \leqq t_2 \leqq t_3. \tag{16}$$

Finally we introduce the following functions:

$$M_0 = L_1 L_2 L_4, \quad M_2 = L_1 L_3 L_4, \quad M_4 = L_1 L_5 L_2, \quad M_6 = L_1 L_3 L_5. \tag{17}$$

Analogous to (16) we have

$$M_i(t_1, t_2) M_i(t_2, t_3) = M_i(t_1, t_3) \text{ if } t_1 \leqq t_2 \leqq t_3 \quad (i = 0, 2, 4, 6). \tag{18}$$

$M_i(t_1, t_2)$ is the probability that an individual who is in the state S_i at t_1, is still in S_i at t_2 $(t_1 \leqq t_2,\ i = 0, 2, 4, 6)$. The functions M can therefore be called "survival functions." With the help of these functions, solutions to the Markov process are readily obtained. Obviously we have

$$P_0(t) = M_0(0, t). \tag{19}$$

The probability that the individual is in S_2 at t and made the transition $S_0 \rightarrow S_2$ in the time interval $(u, u + du)$ is $P_2(t) p_2(u \mid t)\,du + o(du)$. On the other hand, for this to happen the individual has to stay in S_0 until u, which happens with probability $M_0(0, u)$; then he has to make the transition, which happens with probability $q_2(u)\,du + o(du)$; and then he has to stay in S_2, which happens with probability $M_2(u, t) + o(1)$. This leads to the equation

$$P_2(t) p_2(u \mid t) = M_0(0, u) q_2(u) M_2(u, t). \tag{20}$$

Similarly we obtain

$$P_4(t) p_4(v \mid t) = M_0(0, v) q_4(v) M_4(v, t). \tag{21}$$

$$\begin{aligned} P_6(t) p_6(u, v \mid t) &= M_0(0, u) q_2(u) M_2(u, v) q_4(v) M_6(v, t) \text{ if } u < v \\ &= M_0(0, v) q_4(v) M_4(v, u) q_2(u) M_6(u, t) \text{ if } u > v. \end{aligned} \tag{22}$$

$P_2(t)$ is obtained by integrating the right-hand side of (20) with respect to u. Similarly, $P_4(t)$ results from integrating the right-hand side of (21) with respect to v and $P_6(t)$ from integrating the right-hand side of (22) over both u and v, using the first equation in the region $u < v$, the second in the region $u > v$. By using (17) and (18), equation (12) can now easily be established.

The treatment of more general situations, for example, the case when A and B are not completely independent, follows the same lines as illustrated in the example just given.

If one would only be interested in deriving equations (12), (13) and (14) in the case of complete independence, then it is possible to do this by an elegant method, suggested by Mr. J. Karush (private communication), which obviates the necessity of deriving first explicit expression for the $P_i(t)$. For simplicity we shall give the derivation for the process represented in figure 1, but the type of proof can equally well be used for more complicated processes, such as the one represented by figure 2. If A and B are completely independent, then the resulting Markov process is the superposition of three completely independent processes, which we shall denote by I, II and III: I is a Markov process with two states: a live and a dead state, and a transition intensity $q_1(t)$ from one to the other. II is a Markov process with three states: alive without disease A, alive with A, and dead from A; with transition intensities $q_2(t)$ and $q_3(t,u)$ respectively. III is a Markov process with three states: alive without B, alive with B, and dead from B; with transition intensities $q_4(t)$ and $q_5(t,v)$. Let the random variable T_1 be the transition time in process I. Similarly, let U and T_2 be the two transition times in II, V and T_3 in III. We have then that T_1, (U,T_2) and (V,T_3) are completely independent. Let S_a denote the state of being alive, S_A the state of being alive with A (B may or may not be present), S_B the state of being alive with B (with or without A), and S_{AB} the state of being alive with both A and B, all at time t. Thus, S_{AB} coincides with S_6. We have

$$P\{S_A \mid S_a\} = P\{U < t \mid T_1 > t, T_2 > t, T_3 > t\} = P\{U < t \mid T_2 > t\}$$

by independence. Similarly, $P\{S_B \mid S_a\} = P\{V < t \mid T_3 > t\}$. Finally,

$$\begin{aligned} P\{S_{AB} \mid S_a\} &= P\{U < t, V < t \mid T_1 > t, T_2 > t, T_3 > t\} \\ &= P\{U < t \mid T_2 > t\} P\{V < t \mid T_3 > t\} = P\{S_A \mid S_a\} P\{S_B \mid S_a\}. \end{aligned}$$

In words: given that an individual is alive at t, the property of having A is independent of the property of having B. From this, (12) follows easily. It is also possible to write down expressions for the $P_i(t)$ in terms of the random variables introduced above. For example: $P_2(t) = P\{T_1 > t\} \cdot P\{U < t, T_2 > t\} P\{V > t\}$. This also leads immediately to (12).

SUMMARY

The paper is concerned with a description and analysis of two different methods of defining the concept of association between two diseases A and B. The first method (section 2) is based on the classification of a well-defined group of individuals into four categories, according to their A-B disease pattern, *i. e*, those who have neither A nor B, those who have A only, those who have B only, and those who have both A and B. Different choices for this group of individuals may lead to contradictory conclusions as regards to association. The second method of definition of association (section 3) is based on a mathematical model of the phenomenon of acquiring diseases A and B and succumbing to them. The second method seems to be more meaningful than the first. A few simple mathematical models—both deterministic and stochastic—are presented, and some consequences drawn. The mathematical treatment of the stochastic models is included in the Appendix.

Two concepts have to be distinguished: incidence-independence and mortality-independence. It is shown in sections 4 and 5 that in order to assess completely the types and amount of dependence between A and B, it is not sufficient to compute a simple cross ratio from the classification of individuals into 4 categories according to their A-B sickness pattern. Some suggestions are made regarding the types of observations which would have to be made in order to answer all questions pertinent to the association between A and B.

ACKNOWLEDGMENTS

The author is highly indebted to all those participants in the Research Seminar on Statistical Problems in Health whose thoughts and suggestions have been incorporated in this paper, especially Drs. J. Neyman, D. R. Cox, J. Karush, Agnes Berger, Elizabeth L. Scott and W. Smith.

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BOOK REVIEWS

Anthropology and Human Nature. By M. F. Ashley Montagu. 390pp. Porter Sargent, Boston, 1957. $6.00.

I have a friend who runs a restaurant, quite successfully so. The secret of his success, he says, is a wise use of left-overs. In an analogous sense Prof. Montagu is also successful as a purveyor of literary comestibles. This volume is another of the very palatable and digestible *ragout à la Montagu,* a specialty of the author. To those of us who keep pace with his considerable output there is a lot of rehashing in the concoction. Don't get me wrong! I'm all for this sort of thing. However, the element of surprise is not in content, per se, but in the way the volume is put together.

No far-seeing social scientist will disagree with the main theme of the book: to put into operation within the cultural framework the findings of social science, so that the social scientist is in effect a "social engineer." To close the gap between theory and practice is a defensible aim. The real issue is the definition and elucidation of a "finding" as fact rather than as opinion. Montagu, to judge by his very extensive bibliographies, reads widely. He has a flair for synthesis and restatement that cannot be denied. Personally, I learn a lot from what he brings together. But I'm not always sure, or clear, if the statements made are evidence or support; evidence in the sense of objective data, support in the sense of subjective (re)-interpretation. I have the feeling—as I have had in his earlier works—that he tries too hard to do away with "race" and substitute "ethnic group." The problem is not simply a pre- or post- genetic viewpoint. It is, I think, a confusion between human groups as biologic units and human groups as socially interacting units—not what Man is, but what men do about it! Montagu's logic seems to be that to do away with "race" is to do away with a major cause of human group tension and competition; this, in turn, bolsters his idea that Man is innately an organism that seeks species survival and, hence, is instinctively against intra-*sapiens* conflict.

In 1946 the late Raymond Pearl (*Man the Animal*) neatly analyzed the relative variability of normal adult human beings. If size and shape dimensions be rated as 1.0, then physiological dimensions = 1.8, weights (alimentation factor) = 3.4, motor and sensory performance = 9.3, judgment = 47.4, and behavior = 135.5. To put it succintly, human behavior is over 100 times more variable than human biology. I see no way in which a substitution of terms will militate against this sort of picture. Competition (or war, if you will) between groups is not a matter of biology. It is one of mental outlook, habits, ways of doing, and so on—the warp and woof of human behavior as packaged into culture. And no fooling around with words will alter this!

The Chapter on " Sexual Development and Sex Beliefs " leans heavily upon his 1946 " Adolescent Sterility " which in 1957 was greatly expanded into " The Reproductive Development of the Female "—another excellent job of restatement plus up-dating and evaluation.

The Chapter on " Crime " finally beats to death the tired old horse of Hooton's *Crime and the Man.*

The book ends with two bibliographies, one of 17 pages pertinent to the data in the book (by other authors), one of 12 pages (equally pertinent) of writings by Montagu.

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Effect of Radiation on Human Heredity. 168 pp. WORLD HEALTH ORGANIZATION. Geneva, Switzerland, 1957. $4.00.

Some ionizing radiation exists, varying in amount in different geographical areas, as a part of man's natural environment. Recent technological developments opened up new, potent, man-made sources of ionizing radiations. The biologists are aware of the potential dangers to the health of the present, and, perhaps even more so, of the future generations. They are engaged in intensive search regarding the probable effects of ionizing radiations on the descendants of individuals and of groups of individuals exposed to increased amounts of such radiations. This scrutiny has been carried on for some time by scientists of different nations.* In view of the community of interest and the fact that some

* Cf. 1) BONE AND RADIOSTRONTIUM. By Arne Engström, Rolf Björnerstedt, Carl-Johan Clemedson and Arne Nelson. 139 pp. John Wiley, New York, 1957. No price indicated. 2) BIOLOGICAL EFFECTS OF WHOLE-BODY GAMMA RADIATION ON HUMAN BEINGS. By Harold O. Davidson. 101 pp. Johns Hopkins Press, Baltimore, 1957. $4.50. 3) THE ATOMIC AGE AND OUR BIOLOGICAL FUTURE. By H. V. Brøndsted. Translated by E. M. Huggard. xiv + 80 pp. Philosophical Library, New York, 1957. $2.75. 4) OUR NUCLEAR ADVENTURE. By D. G. Arnott. xi + 170 pp. Philosophical Library, New York, 1958. $6.00.

radiations can not be limited to the territory of any one nation, it was proper that the subject was taken up by the WHO. An international study group convened in August 1956 at the University of Copenhagen. Its deliberations comprise the present report. Some of the twelve papers deal with various sources of radiation and their possible contribution to genetic damage. Other papers discuss such topics in genetics as effects of inbreeding, estimation of spontaneous mutation rates, and genetic trends in public health.

There is general agreement among the writers that a threshold dose does not exist for genetic damage. In contrast, the organism has the capacity to repair, within limits, the damage resulting from exposure to parts of the body other than the gonads. The ability of the body to recuperate from radiation damage may be illustrated by the following example. Seven hundred *r* (roentgens) of a given quality of x-rays administered in one hour will produce a skin erythema. If the same degree of erythema were to be produced by two doses administered at intervals of 24 hours, each dose would need to be about 535 *r*. Thus a total of 1070 *r* would be necessary to produce the same effect.

The International Commission on Radiological Protection (ICRP), the Medical Research Council of Great Britain (MRC), and the U. S. National Academy of Sciences (NAS) are essentially in agreement on maximum permissible levels of ionizing radiation. They agree that it would be desirable to limit the dose to the gonads of individuals to 5 *r* per year, and to a dose not exceeding 50 *r* from conception to thirty years of age. ICRP recommends that the gonadal dose for the general population should be of the order of background radiation and NAS sets as a maximum permissible gonadal dose 10 *r* to age 30.

It was felt that some radiation sources—such as radioactive waste—will be technologically controlled and that effective measures will be designed to protect those exposed to certain other radiation sources. It was pointed out, however, that accidents and unforseen events may spread materials with a long half-life throughout the general population which would be harmful in hitherto unknown ways.

One author observed that there is no evidence of an increase in the undesirable traits which might be expected should there be an increase in the mutation rate resulting from the expanding use of medical radiology. He emphasized, however, that there may be an increased incidence of leukemia among certain x-ray treated individuals and that this radiation leukemogenesis may be a non-threshold effect analogous to the induction of mutations. It was observed that deaths from leukemia in the general population are on the increase and may be associated with the increased exposure to ionizing radiation.

Lejeune described three criteria of the genetic influence of radiation: (a) frequency of abnormalities; (b) frequency of still births and miscarriages; (c) variation in the sex ratio. Specifically among children of fathers who were radiologists, as compared with the children of fathers in other medical specialities, the increase of congenital heart disease was found to be significantly higher.

One cannot overemphasize the importance of studying the effects of radiation background on man rather than using only data extrapolated from animal studies. Gapal-Ayengar suggested that studies be carried out in such places as Travancore-Cochin-State in India where the background radiation is considerably higher than in other parts of the world. The nearby areas, with normal background radiation, could provide the comparative data for controlled studies of radiation-induced mutations in man.

To study on a large scale the effects of a rising background radiation, a "Family Register Index," with punch cards containing essentially a family tree, was suggested. Births, deaths, marriage data and offspring would be included. If such data were collected over a ten year period, there would be a million infants born to the marriages under study. Approximately half of these would have at least one brother or sister for comparison. Such a study would also provide valuable experience in handling masses of information which would be needed to evaluate genetic trends in the health of the nation.

Permissible levels of exposure to ionizing radiation are too often thought of as levels which are wholly acceptable and may be taken without undue concern. The authors warned of not gearing a radiological health program to currently accepted levels. We must rather continue to evaluate all areas of possible radiation exposure and genetic damage. It must be kept in mind that the future health significance, just as the national debt, must be considered seriously by this generation for the well being of the coming generations.

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Geography in the 20th Century. Edited by GRIFFITH TAYLOR. xi + 674 pp. Philosophical Library, New York. $10.00.

"When this book was reprinted in 1953 various small revisions and additions were made to the text and two new chapters, XXVI ('Cartography') and XXVII ('The Geographical Interpretation of Air Photography') were added. For the 1957 edition, one new chapter, XXVIII ('The Relations of Geography and History'), has been added and Chapter XIII ('Exploration of Antarctica') brought up to date." So reads the Note to 1957 Edition. For a book with such a time-spanning title, it is difficult to see why the present re-publication merits consideration as an edition, for it is as markedly early twentieth century in its outlook as the original volume.

Chapters by such forward-looking authors as Bowman ("Settlement by the Modern Pioneer"), Peltzer ("Geography and the Tropics") do something to give the work a more constructive outlook, but description, argument as to the nature of geography and its subdivisions, and a deterministic acceptance of environmental dominance still crowd the pages and overshadow the search for basic principles, the scientific sifting of evidence, and the pursuit of order which so increasingly characterize the twentieth century.

To be sure, the chapter on "Environmentalism and Possibilism" strikes out for the liberal attitude in championing those who "without denying the limits every environment sets to man's ambition . . . emphasize the scope of man's action rather than these limits." But even this liberalism apparently sits heavily on the editor's spirit, to judge by the rather cutting interjections he makes as footnotes, notwithstanding that it has almost become conservatism in an age where man confidently expects to break the last remaining tie with his terrestrial environment.

It has been said that no one should become a geographer until he has first excelled in some other branch of science. If geography is to include man in its comparisons of situations at different points on the earth's surface, then a sprinkling of geographers should be drawn from competent human biologists. Until that happens, there seems little chance that geographic texts will sufficiently recognize the limitations of the survey method, the non sequiturs of correlation, and the inconsequence of speculation in the absence of evidence; or that the salutary effects of controlled experiment will be brought to bear upon the biological issues.

Biology was lifted out of the descriptive phase by the genius of

Darwin and Wallace, who saw underlying principles where others saw only cases. Geography badly needs a Darwin!

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The Pharmacology of Psychotomimetic and Psychotherapeutic Drugs. Ed. by S. S. Kety. Annals of the New York Academy of Sciences, *66,* 417-840, 1957.

Techniques for the Study of Behavioral Effects of Drugs. Ed. by P. B. Dews. Annals of the New York Academy of Sciences, *65,* 247-356, 1956.

The Neurohypophysis. Ed. by H. Heller. xv + 275 pp. Butterworth Scientific Publications, London, 1957. $7.00.

In fairness to authors and publishers a statement concerning the specific merits of the three books must be made before I attempt to consider in a more general manner the problem raised by these and closely related works.*

The volume *The Pharmacology of Psychomimetic and Phychotherapeutic Drugs* brilliantly organized by S. S. Kety deals with the action of drugs which, like lysergic acid, mimic symptoms occurring in mental disease and those which, like the tranquillizers, are used for therapeutic purposes. The clinical experiences are described briefly whereas the experimental work is reviewed in considerable detail from the point of view of the neurophysiologist, biochemist and psychologist. The section on serotonin and its relation to mental diseases makes fascinating reading. Among the neurophysiological papers the essay of H. Grundfest on bioelectrical phenomena is of considerable value beyond the specific purposes of this monograph.

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* Cf. 1) Psychotropic Drugs (Proceedings of the International Symposium on Psychotropic Drugs, Milan, May 9-11, 1957). Ed. by S. Garattini and V. Ghetti. xiv + 606 pp. Elsevier Publ. Co., Amsterdam, 1957. $19.50. Distributed by D. Van Nostrand Co., Princeton, N. J. One section is devoted to behavioral effects. 2) Neuropharmacology. Edited by Harold A. Abramson. 381 pp. Josiah Macy, Jr. Foundation, New York, 1957. $4.50. 3) Psychopharmacology. Edited by Harry H. Pennes. xiii + 362 pp. Hoeber-Harper, New York, 1958. $8.00. 4) The Relation of Psychiatry to Pharmacology. By Abraham Wikler. viii + 322 pp. Williams & Wilkins, Baltimore, 1957. $4.00. 5) Pharmacology in Medicine. Edited by Victor A. Drill. xi + 1273 pp. McGraw-Hill, New York, 1958. $19.50. Part seven (pp. 309-334) on "Drugs Affecting Behavior" was contributed by Peter B. Dews.

The *Techniques for the Study of Behavioral Effects of Drugs* is a misnomer insofar as the book covers not only techniques but also, in some detail, the action of these drugs. It supplements (and overlaps in part with) the previous volume which in one section is concerned with the behavioral effects of drugs. In the " schedule-oriented operant technique " developed by Skinner and his school the rat is trained to press a bar to obtain food. The performance (rate of pressing) depends on the schedule of reinforcement which is varied widely. The ingenious recording technique permits the accumulation of enormous quantities of data and the demonstration of the effects of various drugs on this performance. This technique has been combined with sensory discrimination in the pigeon in which the pecking response is utilized (Blough). N. E. Miller advocates and practises the application of several behavioral measures in order to avoid the pitfalls due to the exclusive usage of a single response. The development of this branch of research and its application to the problem of behavior and mental disease has been so rapid that some caution seems advised as expressed by M. Sidman in this symposium, who believes that " the discovery of relations between behavior and other biological phenomena will lead inevitably to the elimination of a great deal of psychopharmacologic investigation that now seems exciting but is actually little more than aimless wandering when compared to future potentialities in this field."

The third volume *The Neurohypophysis* edited by H. Heller is likewise a symposium. Its scientific level is very high, and the point of view brought to bear on the neurohypophysis is very broad. Anatomical (including comparative anatomy), physiological, pharmacological and biochemical investigations are reported. This work is highly recommended to those interested in neuro-endocrine relations.

The rapid pace of recent scientific advancements accounted for, in part, by large scale government support has led to a considerable increase in the number of published symposia. Their value for the scientific worker is obvious, but Bertrand Russell's remark: " If a book is to have value except for a work of reference it must be the work of one mind " should not be forgotten. Only then synthesis can be achieved in problems which like those dealt with in these three symposia are obviously concerned with organismic functions of highest complexity.

The great appeal which psychopharmacology exerts on investigators lies not only in its new approach to the problem of mental illness but also in the fact that these drugs permit the normal individual to observe in himself alterations in "psychic" functions leading to experiences which Osmond describes as "most strange, most awesome, and among the most beautiful things in a varied and fortunate life. These are not escapes from but enlargements, burgeonings of reality."

The question arises now whether the alterations of psychic functions by psychomimetic and psychotherapeutic drugs are understandable within the framework of neurophysiological concepts which seem to account, to some degree, for such fundamental psychic processes as emotion and perception. The previous work of the writer (*Physiological Foundations of Neurology and Psychiatry*, 1953, and *Autonomic Imbalance and the Hypothalamus: Implications for Physiology, Medicine, Psychology, and Neuropsychiatry*, 1957, both published by the University of Minnesato Press) suggests that:

1. Emotional excitement is associated with an excitation of the hypothalamus. Its posterior division discharges "downward" and activates the somatic and particularly the sympathetic division of the autonomic nervous system.

2. An "upward" discharge from the hypothalamus influences the cerebral cortex as a whole. This discharge interacts in the specific sensory projection areas with sensory impulses from eye, ear, etc. This interaction is essential for perception.

3. Changes in cortical functions alter in turn the degree of excitability of the hypothalamus and thereby the intensity (and also the quality †) of the hypothalamic-cortical discharge.

4. Changes in the excitability of the posterior hypothalamus are associated with corresponding changes in the intensity of the upward- and downward discharges.

Applied to the problem of psychopharmacological action one would expect that drugs acting on the hypothalamus directly or indirectly (via the cortex) would produce parallel changes in psychic and autonomic processes (i. e., in up-and downward discharges). It is interesting to note that several drugs alter autonomic and psychic events in the expected manner. Thus lysergic acid (LSD) causes pronounced excitatory psychic effects and sympathetic discharges, whereas 2-brom LSD is without effect on the sympathetic system and the psyche (Rothlin). Another psychomimetic drug (mescaline) is likewise activating the sympathetic system, whereas reserpine depresses psychic functions and diminishes sympathetic discharges.

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† Concerning the qualitative changes see the above cited monograph (1957) and my paper Acta Physiol. Pharmacol. Neerland. 6: 111, 1957.

Thus a relatively simple scheme brings some coherence into the mass of data obtained in different species while normal and abnormal " psychic " processes occurred. It remains to be seen whether this scheme will be confirmed by, and helpful in, future investigations.

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An Appraisal of the Longitudinal Approach to Studies of Growth and Development. By DANKWARD KODLIN and DONOVAN J. THOMPSON. 47 pp. Purdue University, Lafayette, Indiana. Monographs of the Society for Child Development, Serial No. 67, 1958. No price indicated.

In the early years of the present century, students of growth repeatedly emphasized the importance of longitudinal studies in which each subject is measured repeatedly over a period of time. As a result, several such studies were set up, first in the United States and later elsewhere. Some of these studies have now accumulated information on many individuals over a period extending from early childhood to adulthood. The problems, both practical and statistical, of reducing this mass of information into a coherent story are acute and progress in solving them has sometimes seemed disappointing. It is not surprising therefore that a certain reaction against an uncritical dependence on longitudinal data should have set in, and the Apprasial seems to have originated in this way. It claims to answer the tough question " When are the longitudinal and the cross-sectional approaches interchangeable, if at all? "

The first half of the pamphlet presents a list of criteria—all fairly obvious—by which an investigator can decide whether his problem necessitates a longitudinal study, followed by a very brief historical review and a description of the programs in operation at the Harvard University School of Public Health, the Fels Research Institute and the Child Research Council, Denver. The comments on these studies emphasize the "pernicious results of long-term accumulation of data"—collector's instinct has too often led to the collection of figures whose ultimate purpose is never adequately considered, a criticism which should by no means be confined to longitudinal studies.

The second part of the monograph compares methods of analysis of cross-sectional and longitudinal data and their efficiencies in various respects. A good deal of needlessly elaborate mathematics is used to establish some rather obvious conclusions, which amount in summary to saying that the results of cross-sectional and longitudinal studies in many aspects agree, apart from sampling errors. When sampling considerations are introduced, the authors discuss longitudinal studies in which subjects enter and leave during the course of the program; they reproduce the formulae from Cochran's textbook without considering too closely their appropriateness in the present context. No mention is made of the basic paper on this problem by Patterson (J.Roy.Statist. Soc.B,. *12*, 241, 1950) and only passing reference is made to Tanner's detailed exposition of Patterson's results in anthropometric terms (Hum. Biol., *23*, 93, 1951). An italicized conclusion reads "Completely longitudinal records are of no advantage over cross-sectional ones if they are only used to estimate means at particular occasions, and 'mean growth curves' established in either way are equally informative." The first part of this sentence is indeed unexceptable—nobody supposes that longitudinal studies are needed to establish distance standards—but the second part is wildly misleading; the sentence quoted stands over a table showing that a longitudinal sample will often be 10 to 20 times as efficient as a cross-sectional sample in assessing mean growth over a single interval. Shuttleworth's classic monograph of 1937 in the same series seems to have made no impression on the authors!

The present status of longitudinal studies is far from satisfactory, and the authors have done a service by insisting on the necessity of clear aims, continuous working-up of results and scepticism in relation to non-random samples; but an adequate appraisal of the longitudinal approach, with full consideration of its many problems, remains to be written.

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M. J. R. HEALY

BOOK NOTICES

PRINCIPLES OF PERFECTION. By S. Howard Bartley. xii + 482 pp. Harper, New York, 1958. $6.50. College textbook by a specialist in the study of vision, presenting psychology as a biological science and "meant to be a decided step in getting away from the common persistent retention of mentalistic concepts and usages, while at the same time attempting to recognize the role of awareness (experience, consciousness, etc.)."

SCIENTIFIC SOCIETIES IN THE UNITED STATES. By Ralph S. Bates. ix + 297 pp. Columbia University Press, New York, 1958. $6.50. The text of the useful first edition (1945), dealing with the history and the influence of the scientific societies in the U. S., was supplemented by a new chapter covering the changes that took place in the intervening years (The Atomic Age, 1945-1955). The bibliography (pp. 225-268) was revised and enlarged. Comprehensive index (pp. 269-297).

RECENT ADVANCES IN OBSTETRICS AND GYNAECOLOGY. By Aleck W. Bourne. vii + 348 pp. Little, Brown & Co., Boston, 1958. $8.00. Among the 13 chapters constituting the 9th edition of the *Advances*, "The Obstetric Pelvis," especially the section on pelvimetry, may be noted.

HIGH-TALENT MANPOWER FOR SCIENCE AND INDUSTRY. By Douglas Brown and Frederick Harbison. 97 pp. Princeton University, Princeton, N. J., 1957. $3.00. "Creative imagination is a gift of but a few in any society. It must be encouraged by understanding support and satisfying use. (It can not be bought at a price but must be developed) through the systematic improvement of a whole complex of cultural, educational, and industrial institutions and processes." The two essays appraise relevant policies at home and abroad.

INTRODUCTION TO THE THEORY OF DETERMINANTS AND MATRICES. By Edward Tankard Browne. xi + 270 pp. University of North Carolina Press, Chapel Hill, 1958. $7.50. Attempting to avoid an excessively abstract approach the author presents the fundamental facts, together with proofs and illustrations, keeping in mind not only students of mathematics but also the students from "foreign fields."

MULTIVARIATE CORRELATIONAL ANALYSIS. By Philip H. DuBois. xv + 202 pp. Harper & Brothers, New York, 1957. $4.50. Though written with special reference to social science research, the presentation will be found useful by workers in human biology who frequently observe and experiment within multivariate contexts. Both multiple correlation and multiple "factor analysis" are considered.

MINNESOTA STUDIES IN THE PHILOSOPHY OF SCIENCE. Vol. II. Edited by Herbert Feigl, Michael Scriven and Grover Maxwell. xv + 553 pp. University of Minnesota Press, Minneapolis, 1958. $7.00. The volume presents further results of work at the Minnesota Center for the Philosophy of Science on philosophical and methodological problems of science, with special reference to psychology. Feigl's paper on the "mental" and the "physical" stands out on account of its size (pp. 370-497) and thoroughness of analysis.

SCHEDULES OF REINFORCEMENT. By C. B. Ferster and B. F. Skinner. 741 pp. Appleton-Century-Crofts, New York, 1-957. $6.50. "When an organism acts upon the environment in which it lives, it changes that environment in ways which often affect the organism itself. Some of these changes are . . . "reinforcers": they increase the likelihood that the organism will behave in the same way again." This is a detailed report on the effect of varying the ways in which the reinforcements are scheduled. Most of the data refer to pigeons' pecking a key on the wall of an experimental chamber but the methodology has important applications for the analysis of the control of human behavior. A glossary is appended.

ANCIENT AND MODERN MAN IN SOUTHWESTERN ASIA. By Henry Field. xiii + 342 pp. University of Miami Press, Coral Gables, Fla., 1956. No price indicated. Based on 3 decades of library research and field work, the volume summarizes present knowledge on 12 areas (Sinai, Israel, Jordan, Lebanon, Syria, Turkey, Caucasus, Iraq, Arabian Penninsula, Iran, Afghanistan, and Baluchistan), considering 1) geography (incl. climate, water supply, vegetation, fauna, natural resources), 2) ancient man, and 3) modern man. Appendixes contain extensive anthropometric data (especially stature and cranial measurements and indices).

BIBLIOGRAPHY ON SOUTHWESTERN ASIA: IV. By Henry Field. xlvi + 464 pp. University of Miami Press, Coral Gables, Fla., 1957. No price indicated. This is the fourth in a series of specialized bibliographies covering the region indicated in the title (from Egypt to Indus Valley in West Pakistan, and from Anatolia and the Caucasus to the Arabian Sea). Emphasis is placed on anthropogeography (over 8,000 titles, listed alphabetically by author). There is an author index but no subject index.

HUMAN TYPES. By Raymond Firth. vi + 224 pp. Barnes & Noble, Inc., New York, 1957. $2.25. Revised edition of an introduction to social anthropology, first published in 1938. The first chapter deals with racial traits and mental differences.

MINERAL NUTRITION AND THE BALANCE OF LIFE. By Frank A. Gilbert. xv + 350 pp. University of Oklahoma Press, Norman, 1957. $5.95. Designed to supplement the author's "Mineral Nutrition of Plants and Animals" (1948). Includes subject matter not originally covered, such as the history and geographical distribution of the deficiencies of individual minerals. In the body of the text, human nutrition is treated only incidentally (especially in connection with iodine). The last chapter (pp. 256-263) considers briefly some of the implications of mineral nutrition for man.

ATOMIC ENERGY IN MEDICINE. By K. E. Halnan. ix + 157 pp. Philosophical Library, New York, 1957. No price indicated. A readable introduction to a rapidly expanding field.

TUBERCULOSIS IN WHITE AND NEGRO CHILDREN. Vol. I. By Janet B. Hardy. vii + 122 pp. Vol. II. By Miriam E. Brailey. 103 pp. Harvard University Press, Cambridge, 1958. $7.50 and $4.50. Volume I presents the roentgenological, Volume II the epidemiological aspects (prognosis of tuberculosis infection, the risk of developing reinfection) of a study originated in Baltimore in a special outpatient clinic in 1928, with follow-ups continued to 1950. The mean annual reinfection rate among Negro children was ten times the rate noted for white children.

CLIMATOLOGY. By W. G. Kendrew. xv + 400 pp. Oxford University Press, New York, 1957. 42 s. Protective devices available to modern civilized man tend to obscure the fact that "climate is the most fundamental and far-reaching of the natural elements which control life." The book, the first edition of which appeared in 1949, deals with temperature, atmospheric pressure and winds, water in the atmosphere and its condensations and climatic types.

INITIATION AUX MÉTHODES STATISTIQUES EN BIOLOGIE. By Maxime Lamotte. 144 pp. Masson & Cie, Paris, 1957. 2,000 fr. An introductory text by a professor of zoology at the Faculty of Sciences (Paris).

OBJECTIVE TESTS AS INSTRUMENTS OF PSYCHOLOGICAL THEORY. By Jane Loevinger. 59 pp. Psychological Reports, Missoula, Mont., 1957. $2.50. A thoughtful monograph centered around the concept of "construct validity" of psychological tests, defined in terms of content, internal structure and relation to outside variables. The author stresses the need for the recognition of psychometrics as the handmaiden of psychology rather than merely of psychotechnology.

SYSTEMATIC SOCIOLOGY. By Karl Mannheim. Edited by J. S. Eros and W. A. C. Stewart. xxx + 169pp. Philosophical Library, New York, 1957. $6.00. The post-humous edition of "An introduction to the study of society" consists of four parts dealing, respectively, with man's psychological equipment (with emphasis on the flexibility of behavior), elementary social processes, social integration, and factors which make for social stability and change.

A HISTORY OF NUTRITION. By Elmer Verner McCollum. x + 451 pp. Houghton Mifflin, Boston, 1957. $6.00. Covers, approximately, the period 1740-1940 and considers "the efforts of the apothecary type of chemists to learn about the constituents of plant materials; of physiologists who made the early digestion studies; of the occasional penetrating studies of chemists which brought order out of chaos in comprehending the chemical phenomena of the organic kingdom of nature; and also of the feeding studies of the animal husbandry type of investigator, and of the progressively well-controlled studies on men and animals, which made the science of nutrition."

BASIC NUTRITION. By E. W. McHenry. xv + 389 pp. J. B. Lippincott, Philadelphia, 1957. $6.00. A college textbook, growing out of 30 years of experience in research and teaching in the field of nutrition. The author is professor of public health nutrition at the University of Toronto.

CHEMOTHERAPY AND THE CENTRAL NERVOUS SYSTEM. By Henry McIlwain. viii + 328 pp. Little, Brown and Company, Boston, 1957. $10.00. In recent years rapid, almost precipitous developments took place in the chemotherapeutic approach to mental disorders. The author attempted to place them in proper historical perspective and portray them in a wider setting. "There is thus recounted a subject to which chemistry, biochemistry, pharmacology, physiology and clinical medicine have contributed, and in its presentation the interests of workers and students in each of these subjects has been borne in mind."

CANCER STATISTICS FOR ENGLAND AND WALES 1901-1955. By Alan McKenzie. iv + 99 pp. British Information Services, New York, 1957. $1.04. The core of the volume is the tabular presentation of data relating to cancer at various sites for the period 1901-1955. Secular changes in mortality, cancer in children, regional differences, and survival rates from cancer are considered.

PSYCHOBIOLOGY. By Adolf Meyer. Compiled and edited by Eunice E. Winters and Anna Mae Bowers. xxiii + 257 pp. Charles C. Thomas, Springfield, Ill., 1957. $6.50. The lectures, by an outstanding psychiatrist and thinker, delivered in 1931, are grouped in three sections labelled "Psychobiology" (pp. 3-110), Pathology, and Therapy. The Appendix (pp. 187-214) contains a welcome dictionary of Meyer's highly original terminology. Psychobiological data are described in terms of "performances," of integrated processes which are at the same time "physical" and "mental." Psychobiology studies organisms behaving with evidences of meaning.

HIGHWAY RESEARCH BOARD BULLETIN 163: NIGHT VISIBILITY. iii + 32 pp. National Academy of Sciences—National Research Council, Washington, D. C. 1957. No price indicated. Four papers, presented at the Board's 36th annual meeting, deal with roadway visibility, highway lighting, safe stopping distance, and visibility of license plates.

BASIC RESEARCH: A NATIONAL RESOURCE. viii + 64 pp. National Science Foundation, Washington, D. C., 1957. $.45. Prepared and transmitted to the President of the United States in the interest of achieving desirable balance between basic research—a continuing search for new knowledge—and the applications of science to defense, health, and the economy. Portrays the use of basic research, its contemporary status in the U. S. A., and the steps to be taken in strengthening the appeal and opportunities for exploring the unknown.

FEDERAL GRANTS AND CONTRACTS FOR UNCLASSIFIED RESEARCH IN THE LIFE SCIENCES. v + 241 pp. National Science Foundation, Washington, D. C., 1957. No price indicated. The fourth annual report covers the fiscal year 1956. The projects are listed under one of the 20 categories (such as "Structural Biology," "Methodology,'" and "Equipment Design"), and subdivided by state and institution. There is no overall subject or name index which would facilitate orientation.

THE ORIGIN OF LIFE ON THE EARTH. By A. I. Oparin. Translated from the Russian by Ann Synge. xviii + 495 pp. Academic Press, New York,

1957. $6.80. The topic has been of active concern to the author for well over three decades, his first book on "Origin of Life" having appeared in 1924. The expanded presentation was translated into English by Professor Sergius Morgulis (Macmillan, 1938; Dover, 1953). Now the third, enlarged edition of this thorough, experimentally oriented treatise is being made available in English.

PROGRESS REPORT IN CHEMICAL LITERATURE RETRIEVAL. Edited by Gilbert L. Peakes, Allen Kent and James W. Perry. xii + 217 pp. Interscience Publishers, Inc., New York, 1957. $4.75. Effective utilization of recorded knowledge is becoming as important and challenging task as adding new knowledge. The first volume in a projected series entitled "Advances in Documentation and Library Science" represents the proceedings of two symposia held under the auspices of the Division of Chemical Literature, American Chemical Society.

THE VERTEBRATE VISUAL SYSTEM. By Stephen Polyak. xviii + 1390 pp. University of Chicago Press, Chicago, 1957. $45.00. This outstanding monograph, a sequel to the author's *The Retina* (1941), begins with a detailed historical review of investigations of the eye and of the visual pathways and centers of the brain (pp. 9-203). It deals with the visual system from the anatomical, pathological and biological point of view, with consideration of its role in the development of man. Numerous illustrations, extensive bibliography (pp. 1059-1358), and detailed index.

PSYCHOLOGICAL DEVELOPMENT THROUGH THE LIFE SPAN. By Sidney L. Pressey and Raymond G. Kuhlen. xxiii + 654 pp. Harper & Brothers, New York, 1957. $6.00. This may be regarded as a second, substantially revised version of an earlier work, entitled *Life: A psychological survey* (1939), presenting developmental psychology in the framework of the total life span, with emphasis on adulthood and old age. The intervening 20 years increased the need for a comprehensive developmental perspective. Growth, change and decline in physique is also considered (pp. 27-69).

ADVENTURES IN MEDICAL EDUCATION. By G. Canby Robinson. xii + 338 pp. Harvard University Press, Cambridge, 1957. $5.00. This is "a personal narrative of the great advance of American medicine" during the last half century by one of its active participants, perhaps best known for his concern with the social aspects of medicine (pp. 253-280).

THE HUMAN SUM. Edited by C. H. Rolph. vi + 232 pp. Macmillan, New York, 1957. $3.75. Eleven essays, gathered at the initiative of the (British) Family Planning Association and directed to two goals: first, improving the health and happiness of British families by ensuring that parents have the children they want and avoid unwanted children; second, educating the British public in their responsibilities in regard to the tremendous problem of world population in relation to world resources.

THE ECOLOGY OF MAN. By Paul B. Sears. 61 pp. University of Oregon Press, Eugene, Ore., 1957. No price indicated. Richly illustrated, the volume "attempts to integrate various points of view by showing that the human ecological process is a resultant of the interactions of cultural patterns with the environmental complex—physical, biological, and social."

CAREER OPPORTUNITIES IN BIOLOGY. By Russell B. Stevens. 63 pp. Publication 552, National Academy of Sciences—National Research Council, Washington, D. C., 1957. $1.00. An effective pamphlet written for young people considering the choice of career.

REASON AND CHANCE IN SCIENTIFIC DISCOVERY. By R. Taton. Translated from French by A. J. Pomerans. 171 pp. Philosophical Library, New York, 1957. $10.00. Using an illustrative rather than excessively systematic approach, the author describes the different realms of scientific discovery (mathematics, theoretical sciences, observational and experimental sciences), the principal factors in the genesis of discovery (systematic analysis, flashes of thought, role of chance and of error) and its various aspects, such as the originality of discoveries.

THE AGING OF POPULATIONS AND ITS ECONOMIC AND SOCIAL IMPLICATIONS. vii + 168 pp. United Nations, Department of Economic and Social Affairs, New York, 1956. $1.75. Considers trends (past, present, and future) in the aging of populations, their causes, and their implications. The Appendix contains tabular information on present age composition of 70 countries and on changes observed over varying lengths of time in 26 countries.

ROOTS OF SCIENTIFIC THOUGHT. Edited by Philip P. Wiener and Aaron Noland. x + 677 pp. Basic Books, New York, 1957. $8.00. "The historian of ideas is interested not only in the logical, cumulative development of scientific thought but also in the extralogical components and affliliations with developments in the history of the arts, of social institution, of religion, and of philosophy. He aims to shed light on the role played by scientific ways of thinking in shaping the course of civilization." The book, prepared by editors of the *Journal of the History of Ideas*, consists of selections from the first 18 volumes of the journal.

THE NEW ERA IN MEDICAL RESEARCH: A SURVEY. Conducted by Douglas Williams Associates. 63 pp. Merck & Co., Inc., Rahway, N. J., 1957. No price indicated. The full subtitle, "A survey of the attitudes [concerning medical research] of some leading people in the field" specifies the nature of the report. The need for research talent, the need for basic research, the lack of effective communication within the field—these are some of the points of agreement in a field characterized otherwise by a high diversity of opinion.

HEREDITY AND YOUR LIFE. By A. M. Winchester. xii + 333 pp. Vantage Press, New York, 1956. $5.00. A popular "account of everyday human inheritance."

THE LIFE OF MAMMALS. By J. Z. Young. xv + 820 pp. Oxford University Press, New York, 1957. $10.00. Presentation of mammalian life, intended for students of biology and medicine. Integrates information provided by anatomy, histology, embryology and physiology. The inital chapter is devoted to "The control of living systems" (pp. 1-30) and is built around the idea of the body as a self-controlling machine, an idea which serves as a unifying theme throughout.

INFORMATION FOR CONTRIBUTORS

Human Biology, a record of research, is a quarterly journal devoted to the subject its name implies. It publishes original articles dealing with human genetics, development, growth, reproduction and aging, functional anatomy, bio-anthropology, human ecology, population dynamics, etc.

The original manuscripts, typed double-spaced on one side of 8½ x 11 inch paper in complete and finished form precisely as they are to appear, should be sent—packed flat—to the editor, Dr. Gabriel Lasker, Wayne State University College of Medicine, 1401 Rivard Street, Detroit 7, Michigan. It is desirable that a second copy be included in order to expedite prompt consideration, and that an additional carbon copy and copies of all illustrations should be retained by the author.

Where possible, illustrations should be made for reproduction as line engravings, hence drawn in India ink as boldly as possible. Lettering must remain legible after figures are reduced to fit on 4¼ x 6¾ inch pages (letters 1½ to 2 mm high after reduction are adequate). Figures should be numbered consecutively with Arabic numerals. Tables should be numbered independently from 1 up, and both figures and tables should have captions. When the amount of tabular and illustrative material is judged to be excessive or unusually expensive, authors may be requested to pay the excess cost. Galley proofs will be sent to the author and must be returned promptly; when the author makes substantial departures from copy, the cost will be charged to him. Three copies of an abstract (not to exceed 225 words) should be submitted when the proof is returned. One will be sent to the editor of *Biological Abstracts*.

References in the text to the literature should be made by using the author's name followed by the year of publication: "Studies of Garn and Saalberg ('53) . . ." At the end of the article, under the heading "LITERATURE CITED," full references with titles and inclusive page references should appear (double spaced) in alphabetical order of the authors' names: "GARN, S. M. AND J. H. SAALBERG 1953 Sex and age differences in the composition of the adult leg. Human Biol., *25*: 144-153."

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THE RETARDING EFFECT OF PROTRACTED UNDERNUTRITION ON THE APPEARANCE OF THE POSTNATAL OSSIFICATION CENTERS IN THE HAND AND WRIST[1]

BY SAMUEL DREIZEN,[2] RICHARD M. SNODGRASSE,[3] HAMILTON WEBB-PEPLOE[4] AND TOM D. SPIES[2]

Department of Nutrition and Metabolism, Northwestern University

INTRODUCTION

THE oldest and most frequent observation of the effect of undernutrition on skeletal development is a failure to attain expected height. For years, experts have differed in their interpretation of the over-all effect of genetic and nutritional influences on skeletal maturation (Pryor, '36; Sontag and Lipford, '43; Todd, '37; Francis, '39). The advent of roentgenography has permitted important studies of the relation of undernutrition to certain specific features of skeletal development. A prominent aspect readily detectable in serial roentgenograms is

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[1] These studies were supported by grants from the Benjamin & Roberta Russell Educational and Charitable Foundations, Inc., Fuller E. Callaway Foundation, La Grange, Ga., and The Ireland Foundation, Inc.

[2] Northwestern University Medical School.

[3] Temple University, School of Dentistry.

[4] Nutrition Clinic, Hillman Hospital.

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the initial appearance of the postnatal primary and secondary centers of ossification. Todd ('37) and Francis ('39) were among the first to direct attention to the retarding influence of undernutrition on the conversion of the cartilaginous primordium into osseous tissue. Supportive evidence has since been forthcoming from the cross-sectional studies of Abbott, Townsend, French, and Ahmann ('50) in Florida and of Beresowski, and Lundie ('52) in South Africa. A similar impression has been gained in the course of longitudinal studies of approximately 1,000 children at the Nutrition Clinic of the Hillman Hospital, Birmingham, Alabama during the past 17 years. With the accumulation of data derived from a large series of sequential roentgenograms, it is now possible for the first time to make a qualitative and quantitative evaluation of the effect of prolonged undernutrition during infancy and childhood on the onset of ossification in the postnatal centers of the hand and wrist.

MATERIALS AND METHODS

Five hundred and forty-one white, native-born Alabama children of similar ethnic background were arbitrarily selected from the growth study group of the Nutrition Clinic. Serial sequential roentgenograms of the left hand and wrist covering the period during which some or all of the postnatal ossification centers are scheduled to appear were available for each of the 301 boys and 240 girls. Every child was studied carefully and individually including a detailed medical, dietary, family and socio-economic history. Each child selected had evidence consistent with a clinical diagnosis of undernutrition.

The roentgenograms were taken at intervals of three months until a child reached his fifth birthday, and every 6 months thereafter. The size of the series of roentgenograms varied with the age at which the child was accepted for study. Only children with a series in which a reasonably accurate age of appearance of a particular ossification center could be determined were included. When the center appeared between the taking of two roentgenograms, the time of appearance was extrapolated to the nearest month on the basis of the configuration of the center when first seen. The primary ossification centers in the carpus, the secondary ossification centers in the metacarpals, phalanges and distal radius and ulna, and the flexor and adductor sesamoids of the thumb were included in the assessment. The age for the appearance of each of these centers as depicted in the Greulich-Pyle Radiographic Atlas ('50) served as the standard of reference.

OBSERVATIONS

The distributions of the times of appearance of the primary and secondary postnatal ossification centers in the hands and wrists of the undernourished children appear in tables 1 and 2. The frequency of retardation was statistically significant for each center in the boys except those of the adductor sesamoid of the thumb and the distal radius (table 1). The differences between the number delayed and the combined number advanced and on schedule were significant at the 5% level for the capitate and hamate; for all other centers the significance was at the 1% level. In the girls (table 2), statistically significant frequencies of delay were recorded for each center except those of the proximal phalanges II through V and the distal phalanx of the thumb. All but one of the significantly retarded values were at the 1% level; that for distal phalanx III was at the 5% level.

In both the boys and the girls the mean times of appearance for each center were retarded, the delays ranging from 1.2 months (adductor sesamoid of the thumb) to 28.9 months (greater multangular) in the boys and from 1.8 months (proximal phalanx III) to 18.3 months (greater multangular) in the girls. In general, the delay was most accentuated in the carpals (capitate and hamate excluded) followed in decreasing magnitude by the distal ulna, metacarpals, terminal phalanges, middle phalanges, proximal phalanges, and distal radius. Save for the capitate, hamate, and adductor sesamoid of the thumb, the mean extent of retardation in the appearance of each center in the boys greatly exceeded the corresponding values for the girls. For the group, the centers which most closely approximated the Greulich-Pyle ossification schedules were the adductor sesamoid of the thumb in the boys and proximal phalanges II, III and IV in the girls. The comparatively high values for the standard deviations for each center, which frequently exceeded the mean in magnitude, were indicative of a high degree of variability in the time of appearance of the ossification centers in the children comprising this study group.

The number of ossification centers used in the determination of time of appearance in the 541 children ranged from one to 30. In no child was it possible to evaluate all 31 centers usually assessed in the hand skeleton. The mean number of centers per child available for assessment of onset of ossification was 8.6 for the boys and 8.9 for the girls.

The frequencies of the combinations of types of deviation from the Greulich-Pyle standard of the time of appearance of the measurable

TABLE 1

Distribution of the appearance times of the postnatal ossification centers in the hand and wrist of undernourished boys based on the standard time of appearance for each center †

Ossification Center	Number of Cases	Advanced (+) Number	Advanced (+) Per Cent	On Schedule (O) Number	On Schedule (O) Per Cent	Delayed (—) Number	Delayed (—) Per Cent	Mean (Months)	Standard Deviation (Months)
Capitate	33	4	12.12	5	15.15	24*	72.72	—2.7	3.86
Hamate	35	4	11.43	6	17.14	25*	71.42	—4.9	10.71
Triquetrum	87	17	19.54	0	0.00	70**	80.46	—19.0	19.99
Lunate	92	10	10.87	1	1.09	81**	88.04	—26.9	20.89
Navicular	113	5	4.42	3	2.65	105**	92.92	—20.5	15.36
Gtr. Multangular	116	6	5.17	1	0.86	109**	93.96	—28.9	18.02
Lssr. Multangular	115	9	7.83	1	0.87	105**	91.30	—20.2	15.37
Pisiform	122	9	7.37	16	13.11	97**	79.51	—16.6	14.83
Metacarpal I	86	7	8.14	1	1.16	78**	90.69	—15.9	12.52
" II	74	4	5.41	3	4.05	67**	90.54	—10.7	7.57
" III	84	5	5.95	0	0.00	79**	94.04	—12.1	8.86
" IV	82	5	6.09	0	0.00	77*	93.90	—15.1	9.44
" V	75	15	20.00	2	2.67	58**	77.33	—9.7	10.20
Prox. Phalanx I	89	8	8.99	0	0.00	81**	91.01	—15.3	10.74
" " II	76	11	14.47	3	3.95	62**	81.58	—6.6	6.92
" " III	74	9	10.81	7	9.46	58**	78.38	—5.6	6.22
" " IV	76	8	10.52	4	5.26	64**	84.21	—8.0	7.38
" " V	80	6	7.50	5	6.25	69**	86.25	—8.7	7.93
Mid. Phalanx II	81	11	13.58	5	6.17	65**	80.25	—8.7	9.73
" " III	83	8	9.64	5	6.02	70**	84.33	—10.4	9.93
" " IV	83	10	12.05	4	4.82	69**	83.13	—10.4	9.99
" " V	92	11	11.96	6	6.52	75**	81.52	—13.5	13.78
Dist. Phalanx I	80	8	10.00	5	6.25	67**	83.75	—11.7	9.37
" " II	89	14	15.73	5	5.62	70**	78.65	—10.3	11.24
" " III	85	5	5.88	1	1.18	79**	92.94	—15.0	9.87
" " IV	83	5	6.02	1	1.20	77**	92.77	—15.7	10.42
" " V	89	14	15.73	6	6.74	69**	77.52	—10.5	11.49
Adductor Sesamoid	75	26	34.67	14	18.66	35	46.66	—1.2	11.97
Flexor Sesamoid	53	12	22.64	1	1.89	40**	75.46	—7.4	10.74
Distal Radius	70	23	32.85	6	8.57	41	58.57	—5.6	9.03
Distal Ulna	124	14	11.29	0	0.00	110**	88.71	—19.4	16.88

† Greulich-Pyle Standard.

* Significant at 5% level.

** Significant at 1% level.

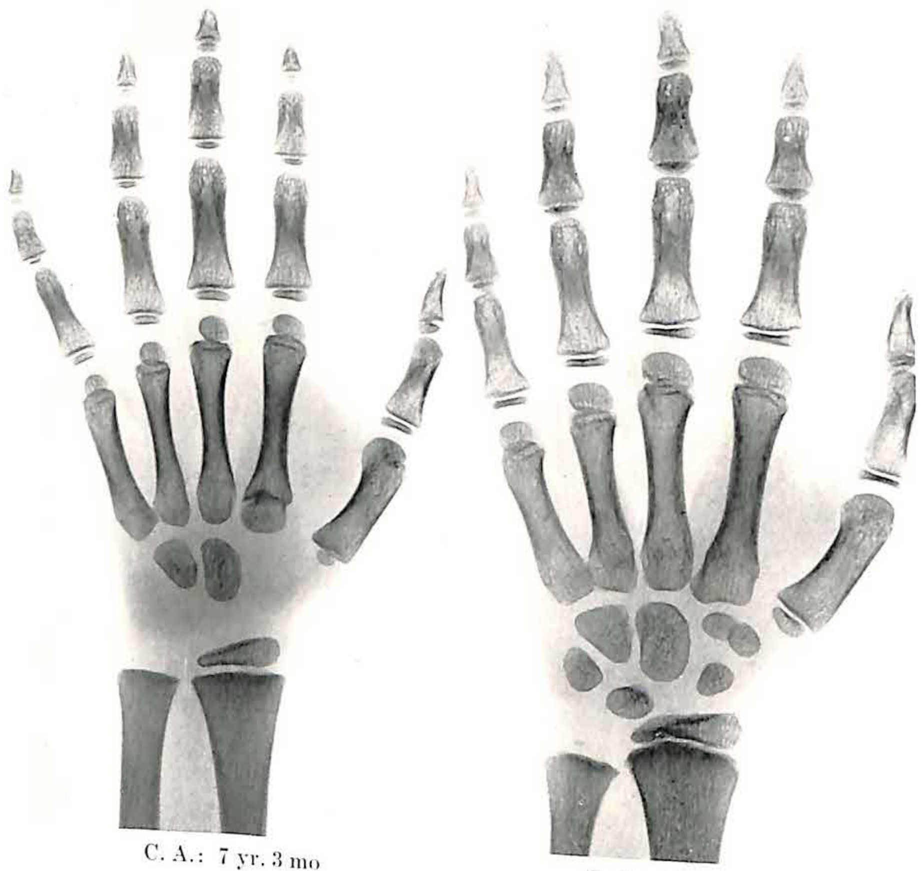
TABLE 2

Distribution of the appearance times of the postnatal ossification centers in the hand and wrist of undernourished girls based on the standard time of appearance for each center †

Ossification Center	Number of Cases	Advanced (+) Number	Advanced (+) Per Cent	On Schedule (O) Number	On Schedule (O) Per Cent	Delayed (—) Number	Delayed (—) Per Cent	Mean (Months)	Standard Deviation (Months)
Capitate	24	1	4.17	4	16.66	19**	79.16	—4.0	3.95
Hamate	24	1	4.17	4	16.66	19**	79.16	—5.0	6.24
Triquetrum	68	9	13.73	0	0.00	59**	86.27	—16.6	16.09
Lunate	84	11	13.09	2	2.38	71**	84.52	—15.4	17.00
Navicular	94	9	9.55	5	5.32	80**	85.11	—18.1	15.74
Gtr. Multangular	97	8	8.25	2	2.06	87**	89.69	—18.3	17.06
Lssr. Multangular	99	7	7.07	9	9.09	83**	83.83	—14.0	12.32
Pisiform	117	15	12.82	10	8.55	92**	78.62	—9.9	11.57
Metacarpal I	68	13	19.11	4	5.88	51**	75.00	—7.7	11.39
" II	57	4	7.02	6	10.53	47**	82.45	—5.1	5.08
" III	60	6	10.00	2	3.33	52**	86.66	—5.5	5.54
" IV	66	9	13.63	4	6.06	53**	80.30	—5.3	5.39
" V	63	7	11.11	5	7.93	51**	80.95	—7.4	8.81
Prox. Phalanx I	66	13	19.70	5	7.58	48**	72.72	—6.9	10.89
" " II	52	10	19.23	11	21.15	31	59.61	—2.3	4.55
" " III	54	12	22.22	13	24.07	29	53.70	—1.8	4.43
" " IV	55	10	18.18	13	23.64	32	58.18	—2.8	4.92
" " V	59	14	23.73	10	16.95	35	59.32	—3.2	7.11
Mid. Phalanx II	64	12	18.75	6	9.37	46**	71.88	—6.2	8.55
" " III	64	12	18.75	7	10.93	45**	70.31	—4.9	6.19
" " IV	65	13	20.00	7	10.77	45**	69.22	—4.9	6.39
" " V	74	11	14.86	3	4.05	60**	81.08	—6.4	10.52
Dist. Phalanx I	56	14	25.00	9	16.07	33	58.92	—3.5	6.68
" " II	73	12	16.43	6	8.22	55**	75.34	—6.8	7.79
" " III	61	18	29.51	2	3.28	41*	67.21	—4.6	8.44
" " IV	62	16	25.81	1	1.61	45**	72.58	—5.0	8.56
" " V	70	8	11.42	2	2.86	60**	85.71	—9.3	8.29
Adductor Sesamoid	83	17	20.48	11	13.25	55**	66.26	—6.9	10.95
Flexor Sesamoid	70	4	5.71	16	22.86	50**	71.43	—11.3	13.03
Distal Radius	53	9	16.98	6	11.32	38**	71.70	—3.4	5.14
Distal Ulna	115	7	6.09	6	5.22	102**	88.69	—15.5	12.14

† Greulich-Pyle Standard.
* Significant at 5% level.
** Significant at 1% level.

postnatal ossification centers in the 541 undernourished children are contained in table 3. These combinations refer to the total number of centers in each child in which the onset of ossification could be identified

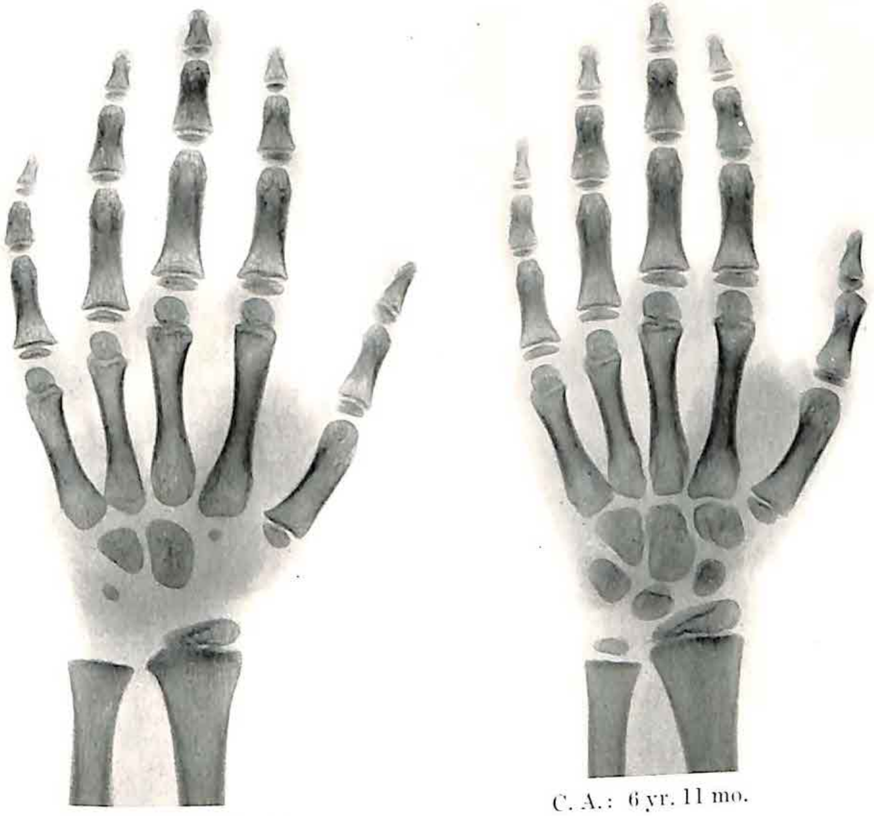


C. A.: 7 yr. 3 mo

C. A.: 7 yr. 4 mo.

Fig. 1. Effect of Known Undernutrition (left) and Known Adequate Nutrition (right) as Shown by Roentgenograms of Hand Skeleton of Two Boys from the Hillman Hospital Nutrition Clinic Series. According to the Greulich-Pyle Standard, Boys with a Skeletal Age of 7 Years Should have 7 Carpal Centers Whereas only Two have Appeared in the Hand of the Undernourished Boy. Well-nourished Boy is Somewhat Ahead of 7 Year Old Standard of Greulich and Pyle. Observe Presence in Hand of Undernourished Boy of Pseudoepiphysis on Distal End of Metacarpal I and on Base of Metacarpal II. Remnants of 7 Transverse Lines of Arrested Growth Near Distal End of Shaft of Radius are Visible in Roentgenogram of this Boy.

in the roentgenograms. Collectively, a delayed appearance of some or all of the identifiable centers was found in 91.3% of the 541 children. In only 29 (5.4%) of the children was the ossification of all detectable centers ahead of schedule and in but three (0.5%) did all appear on schedule.



C. A.: 6 yr. 9 mo.

C. A.: 6 yr. 11 mo.

Fig. 2. Roentgenograms Showing Effect of Known Undernutrition (left) and Known Adequate Nutrition (right) on Hand Skeleton of Two Girls from the Hillman Hospital Nutrition Clinic Series. According to the Greulich-Pyle Standard, Girls with a Skeletal Age of 6 Years 10 Months Should Have 7 Carpal Centers; However, Only 4 are Present in Hand of Undernourished Girl. Note Immaturity of This Hand as Shown by Extent and Contours of Bony Centers When Compared with That of Well-Nourished Girl Who is Advanced Somewhat Beyond Greulich-Pyle Standard.

Quantitatively, the actual amount of deviation in the time of appearance of the postnatal primary and secondary ossification centers in the

TABLE 3

The combinations of advanced, on schedule, and retarded appearance of the postnatal ossification centers in the hand and wrist of 541 undernourished children

	COMBINATION													
	—		—: O		—: +		—: O: +		O		O: +		+	
GROUP	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Boys	162	53.8	35	11.6	40	13.3	40	13.3	2	0.6	3	0.9	19	6.3
Girls	105	43.3	45	18.7	33	13.7	44	18.3	1	0.4	2	0.8	10	4.2
Total	267	49.3	80	14.8	73	13.5	84	15.5	3	0.5	5	0.9	29	5.4

— = Retarded
O = On Schedule
+ = Advanced

hand and wrist of the 541 undernourished children encompassed a range of 116 months. In the boys, the range of deviation extended from 42 months advanced for an adductor sesamoid of the thumb to 92 months delayed for a navicular. The range in the girls extended from 24 months advanced for an adductor sesamoid of the thumb and a lunate respectively, to 65 months delayed for a metacarpal I. In both sexes the carpal centers were the most severely retarded in onset of ossification. In the boys the greatest amount of delay was found most frequently in the greater multangular, with 29.3% of those examined having a handicap exceeding 36 months. In the girls the greatest frequency of retardation in time of appearance in excess of 36 months, which characterized 19.05% of the cases, occurred in the lunate. Among the secondary centers of ossification, that of the distal ulna had the highest frequency in excess of 36 months, the percentages being 14.5 and 4.3 for the boys and girls respectively. Conversely, the least amount of deviation from the standard schedule of appearance for the primary centers of ossification was shown by the capitate in both sexes. None exceeded a delay of 18 months in the boys and 15 months in the girls. The least delay found in the secondary centers was in the proximal phalanx of the third digit in boys (none exceeding 21 months) and in metacarpal IV and the proximal phalanx of the second and fifth digit in the girls (none exceeding 15 months). In both sexes the temporal range of appearance was lowest in the proximal phalanges. In the boys the great bulk of the values fell between plus 9 and minus 24 months; in the girls between plus 9 and minus 18 months. The metacarpals in the girls showed a similar clustering. Of the two long bones of the forearm, the center of the distal ulna in both sexes showed a considerably greater range in time of appearance than did that of the distal radius.

Specific examples of extreme delay in time of appearance of the carpal centers and skeletal maturation, generally, in the 301 boys and 240 girls are presented in figures 1 and 2. The roentgenograms of the undernourished pair are contrasted with those of well-nourished children of the same sex, chronological age, and ethnic origin. The skeletal maturation of the well-nourished children is slightly ahead of the standards contained in the Greulich-Pyle atlas.

DISCUSSION

Analysis of these data supports our previous observations that protracted undernutrition during infancy, childhood, and adolescence affects the maturation of the skeleton. Some of these effects include:

1) a signal failure to attain expected stature (Dreizen *et al.*, '53)
2) a high degree of asymmetry in the maturation of the hand skeleton (Pyle *et al.*, '48)
3) a lagging rate of skeletal development as measured by the Todd inspectional technique (Spies *et al.*, '53; Dreizen *et al.*, '54)
4) a considerable frequency and variety of anomalies in the bones of the hand (Snodgrasse *et al.*, '55a; Snodgrasse *et al.*, '55b)
5) a noteworthy persistance of radio-opaque transverse lines in the distal end of the shaft of the radius (Dreizen *et al.*, '56)
6) a delayed fusion of the epiphyses in the hand, and distal radius and ulna (Dreizen *et al.*, '57).

In the hand and wrist the time of onset of ossification is definitely more variable in the carpals than in the epiphyses (Pyle and Sontag, '43). The advent of ossification is more apt to be delayed by metabolic disturbances in the primary centers of the hand than in the epiphyseal centers (Francis, Werle, and Behm, '39). Francis ('39; '40) reported that inception of ossification in the postnatal centers is materially influenced by the available minerals and vitamins. Recently, the greater susceptibility of the maturation of the carpals than of the other centers in the hand and wrist to interference by intercurrent illness has been re-emphasized (Hewitt, Westropp, and Acheson, '55). The impression that the carpal centers are more variable in time of appearance and more vulnerable to the retarding influence of protacted undernutrition is supported by the observations made on the present series of 541 chlidren. The finding that the capitate and hamate were the least delayed of the carpal centers in time of appearance accords with published reports (Todd, '37; Francis, Werle, and Behm, '39). In the present study, the greater multangular was the most retarded carpal bone in each sex.

Exclusive of the capitate and hamate and the metacarpo-phalangeal sesamoids of the thumb, the primary and secondary centers in the hand were less tardy in time of appearance in girls than in boys. This probably reflects the physiologically more rapid rate of maturation so characteristic of the female sex with the result that adverse environmental circumstances of the same degree of intensity affect males to a greater extent than females. This interpretation is supported by the studies of Greulich ('51; Greulich, Crismon, and Turner, '53) of the growth of Guamanian and Japanese children exposed to unfavorable environmental conditions.

SUMMARY AND CONCLUSIONS

The present study of 301 boys and 240 girls supports and extends our previous inference that protracted undernutrition during the growing period notably affects the postnatal development of the skeleton. Comparison of the roentgenographically detectable advent of ossification in 31 centers of the hand and wrist in the undernourished children with the age standards in the Greulich-Pyle atlas showed that:

1. The mean time of appearance for each of the 31 ossification centers was delayed in every instance in each sex. The mean amount of delay in the boys exceeded that of the girls for all but three centers.
2. The frequency of retardation in onset of ossification surpassed the combined frequency of centers appearing either early or on schedule to a statistically significant degree for 29 and 26 of the 31 centers in the boys and girls respectively.
3. Some or all of the assessable centers were delayed in 504 (91.3%) of the 541 children.
4. Delay in advent of ossification was much more pronounced in the carpals than in the metacarpal and phalangeal epiphyses. In both sexes the delay in the epiphysis of the distal ulna greatly exceeded that of the distal epiphysis of the radius.

5. Quantitatively, in boys the average delay ranged from 1.2 months for the adductor sesamoid of the thumb to 28.9 months for the greater multangular; and in girls, from 1.8 months for the proximal phalanx of the third digit to 18.3 months for the greater multangular.

This clear demonstration of the susceptibility of skeletal maturation to chronic undernutrition once again points to the imperative need for proper and persistent nutritional therapy in the treatment of growth failure resulting from nutritional inadequacy.

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FAT, BODY SIZE AND GROWTH IN THE NEWBORN

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INTRODUCTION

THE size of the infant at birth and the thickness of the subcutaneous fat are obviously correlated variables. Large neonates and post-term infants are not only heavy, but they are well filled out. Small infants, and especially those prematurely born tend to be deficient in superficial fat. Yet within these limits there are many exceptions. The newborn include some long but skinny infants, and others (especially among diabetic progeny) who are excessively fat for their size. The extent of the fat-size relationship is thus a subject for inquiry, and one not touched upon in the literature on fatness (*cf.* Bernhardt, '55).

A second area for investigation is the sex difference in body fat. Girl babies tend to weigh less, but are developmentally more advanced at birth. Boy babies, on the other hand, are both longer and heavier on the average but less advanced developmentally, and notably less hardy. One possibility is that the female neonate has relatively more subcutaneous fat despite her smaller size, as is true in adulthood. Again, it is possible that the male neonate has more subcutaneous fat, a consequence of his greater size at birth. A third possibility is that, sharing a common uterine environment, and equally affected by maternal estrogens that penetrate the placental barrier, neonates of both sexes will tend toward equal superficial fat.

A third problem area concerns regional differences in size and their relationship to stored fat. If the fatter neonate is bigger, as seems to be the case, is he bigger overall, or is the fat-size relationship confined to particular body areas? There is a clinical supposition that the fat new born is disproportionately long in the trunk and relatively short of leg, but this may be an illusion due to the fact that the legs of fat infants look shorter.

In the course of our investigations on growth and development, we have had the opportunity to explore fat-size relationships in a sizeable series of healthy developmentally-mature white infants from a fairly homogeneous population sample. Our data have included not only information on the infants themselves, but a considerable body of data on the mothers, including a number of variables that may relate to individual differences in body fat. It is, therefore, possible to provide new data, not only on relative fatness in the newborn, but also on the determinants and consequences of differences in stored fat.

METHODS AND MATERIALS

The present investigation is primarily based on roentgenogrammetric and anthropometric measurements of 146 developmentally-mature Ohio-born neonates, and 95 mothers from Southwestern Ohio, all regular participants in the Fels Longitudinal Studies. Non-whites, twins and triplets and prematurely-born infants were excluded from the tabulations as were those with known developmental defects. The data thus pertain to a group of neonates with a median estimated length of gestation of 265 days, and a median birth weight of 3,390 gm.

Using the classification of Warner *et al.* ('49) the families involved were principally lower-middle class, with an effective range of upper-lower to upper-middle. Occupationally, nearly 25% of the fathers were engaged in farming and farm-related tasks, and 30% were tradesmen, salesmen and industrial workers. Abaut 40% of the fathers and 60% of the mothers had gone beyond a high school education (*cf.* Crandall and Preston, '56; Sontag, Nelson and Baker, '58). The sample, then consists of semi-urban lower-middle and comparatively well-educated families generally of "old American" ancestry.

Anthropometric data on the infants included birth weight (as measured in the hospital nursery) and the crown-heel diameter, as measured by the Fel pediatrician upon examining the child in the hospital. The remaining long-bone and fat measurements were derived from "birth" x-rays taken at the hospital and contained in the Fels longitudinal files. Anthropometric data on the mothers included stature, weight at the third month of pregnancy, and the last measured weight prior to delivery. From the third month and final pregnancy weight the rate-of-gain was calculated, on an individual basis, by dividing the measured weight gain by the actual number of days intervening. Data on maternal body composition were based on roentgenogrammetric estimates, and by subtraction from the weight in the third month of pregnancy.

To test the representativeness of the sample, anthropometric data on the Fels neonates and the pregnant mothers were compared to data on mothers and infants from a private obstetric practice in Dayton, Ohio, but limiting the obstetric sample to the same range of gestation lengths (median, 265 days; standard deviation, 8 days) and to developmentally-mature singlings. Median values for birth size closely matched in the two samples. Maternal weight at 3 months for the Fels mothers was 59.9 kg, about 3.5 kg higher than in the obstetric group: the 9-month weights were almost identical. Accordingly it was felt that the Fels sample was reasonably representative of the larger local population.

The roentgenogrammetric measurements on the infants were made directly on the antero-posterior "birth" x-rays,[1] which were usually taken on the second day after birth, with a standard deviation of one day. The bony measurements included femoral length (F) and humeral length (H), as shown on figure 1, and spine length (S). Both femoral and humeral length were measured as the maximum extent of calcification along the shaft, but excluding epiphyses where present. Spine length (T_1-L_5) was also taken as a maximum, but without excluding individuals with sacralized lumbar vertebrae or lumbarized sacral vertebrae, because of the still immature state of ossification.

Subcutaneous fat, or more correctly the fat-plus-skin shadow was measured uniformly at the level of the lowest large rib (usually R_{11}), and usually on the right side.[2] Selection of this site was dictated by several factors, one being the fact that the lower limbs were only occasionally included and properly positioned on the birth "torso" x-ray, and the second being the desirability of using a site that could be measured either by pinch-caliper or x-ray on infants and adults alike. Previous studies from this institution have shown the utility of the lower-thoracic (LT) fat site (Garn, '56a, '57b; Garn and Gorman, '56), and the present investigation thus adds to the body of information on LT fat.

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[1] The gonadal exposure incurred in a "birth-torso" x-ray, using high-speed film and 40–50 PKV at 10 milliampere seconds but without gonadal shielding is estimated at 15–30 milliroentgens (.015–.030 *r*) depending on size and sex.

[2] In an initial pilot study using the present material, no systematic difference was found between fat-shadow measurements on the left and right sides respectively. However, in individual cases, where there was evidence of axial rotation, the averaged value $\frac{L+R}{2}$ was employed.

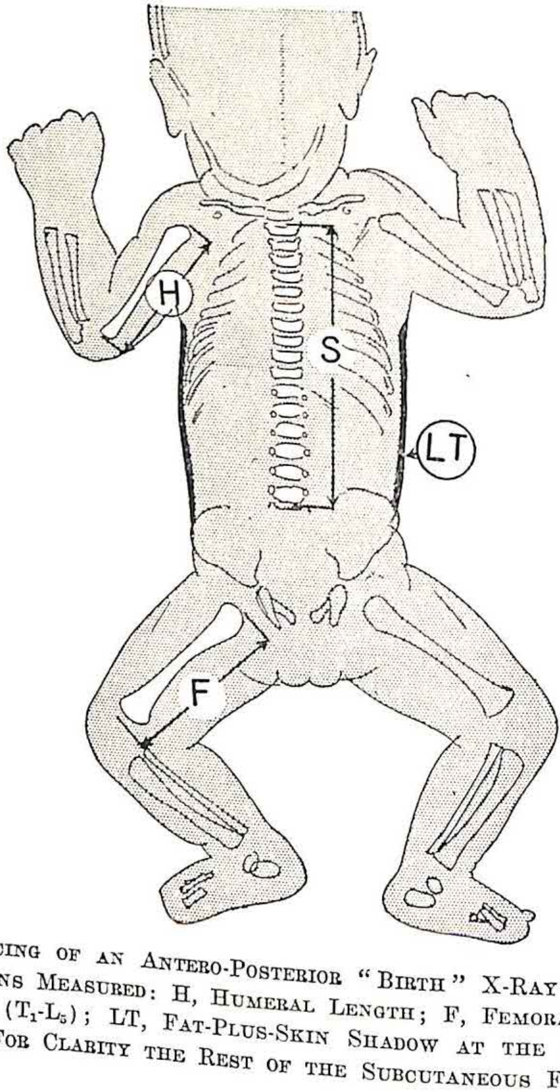

Fig. 1. Tracing of an Antero-Posterior "Birth" X-Ray showing the Four Dimensions Measured: H, Humeral Length; F, Femoral Length; S, Spine Length (T_1-L_5); LT, Fat-Plus-Skin Shadow at the Level of the Lowest Rib. For Clarity the Rest of the Subcutaneous Fat Shadow is Not Shown.

Estimates of the maternal fat-free weight, which were used to further fractionate gross weight into physiologically meaningful compartments, were based on iliac fat measured on x-rays taken subsequent to delivery, using the technique of subtracting the estimated weight of fat from gross weight: the value of the constant *a* was 0.72, as previously described (Garn, '57a). The assumption of a relatively constant fat-free weight was made; this is obviously a limiting assumption but not necessarily an incorrect one (*cf.* Garn, '57a, p. 346).

Since subcutaneous fat and weight were non-normal in distribution, and showed the types of skewness encountered in previous studies, all raw-score distributions were normalized using McCall's technique. The effectiveness of the McCall technique has been investigated by Lacey ('56), and is pictured in figure 20 in Garn and Shamir ('58). Accordingly, the product-moment correlations given in tables 2 through 4 are based on normalized T-scores, and tend to be slightly smaller than comparable correlations using the raw values. However, the fat-weight plot pictured in figure 5 employs raw scores, since it is the purpose of this scattergram to show the fat-weight relationship in normal and in diabetic progeny.

Since the Fels Longitudinal Studies are of the continuing type with constant recruitment, the possibility of secular trends and the further possibility of a changing population sample were considered. However, when the data were sorted according to year of birth, no consistent trend emerged, and there was no evidence that the selection of Fels parents had altered appreciably over the years.

FINDINGS

1. *Fat thickness and sex differences.*

Before attempting data analysis, cumulative frequency distributions for lower thoracic (LT) fat were drawn for boys and girls respectively, and then examined for sex differences and for skewness. As shown in figure 2, considerable skewness (of the type previously encountered for fat distributions) was evident in the fat distributions for both sexes.[3] Accordingly, percentiles were computed, rather than the familiar means and standard deviations, and the median was employed as a measure of central tendency. With one exception indicated in the text, all correlations were then based on normalized T-scores, rather than on the obviously skewed raw scores.

As shown in table 1, where the 5th and 95th percentile range is given, there is a wide span of values for LT fat in the newborn, infants at the

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[3] Pearson's coefficient of skewness (S_K) was 0.58, using the combined-sex cumulative frequency distribution, and calculating the interquartile range by interpolation (*cf.* Arkin and Colton, '56, pp. 40-41).

95th percentile having approximately 2.3 times the thickness of fat of those in the 5th percentile. With regard to sex, however, there appears to be no important fat difference, the medians being almost identical—2.9 and 3.0 mm for boys and girls respectively. The distributions were also quite similar, to the extent that a combined-sex tabulation seemed justified, thus making maximum use of the data on all 146 neonates (*cf.* table 1).

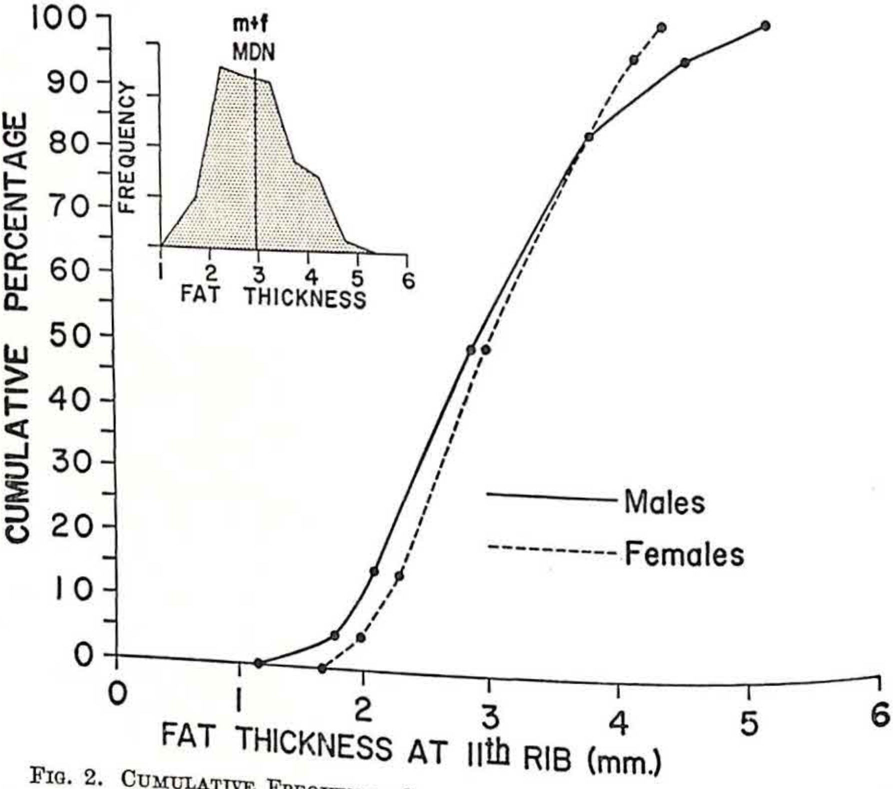

FIG. 2. CUMULATIVE FREQUENCY CURVES FOR LT FAT IN NEWBORN BOYS AND GIRLS. THE TWO CURVES, BOTH OF WHICH ARE SKEWED TO THE RIGHT, DISPLAY LITTLE SEXUAL DIMORPHISM. THE COMBINED-SEX FREQUENCY DISTRIBUTION CURVE IS SHOWN ABOVE.

Despite the virtual identity of fat-thickness values for male and female newborn, it seemed likely that the fat-weight ratios would differ, since the boys tended to weigh more than the girls (boys' Mdn. 3430 gm, girls' Mdn. 3350). Accordingly, the fat-weight ratio, namely fat in mm divided by weight in kg, was computed on an individual basis for the 79 boys and 67 girls respectively. The mean values for the ratio were

TABLE 1

Lower thoracic (Lt) fat percentiles for newborn boys and girls

SEX	No.	FAT PERCENTILES (MM) 5th	15th	50th	85th	95th
Males	79	1.8	2.1	2.9	3.9	4.6
Females	67	2.0	2.3	3.0	3.9	4.2
Combined	146	1.9	2.2	2.9	3.9	4.4

then 0.83 for boys and 0.93 for girls, and the difference (0.10) was significant at better than the 1 per cent level, using Student's *t*-test. While it was true that the sex difference in the ratio was due entirely to the difference in birth weight, it was still evident that the female neonate has a thicker deposit of subcutaneous fat relative to her weight, as is true in later life as well (*cf.* Garn, '57a).

2. *Determinants of birth fat.*

Attention was next directed to a number of variables known to be positively correlated with birth weight, and conceivably related to differences in the degrees of fatness at birth. These included (a) gestation length, since even the small residual variability might loom large in terms of fat deposition, (b) maternal size and weight (big mothers tend to have big babies), and (c) the maternal weight gain. Sibling similarities were also investigated, since these could shed some light on the extent to which subcutaneous fat deposition during late pregnancy is under fetal control. Certain variables, positively and significantly correlated with birth size were not considered however, because their relationship to fatness at birth seemed tenuous. Thus, while paternal length was significantly related to birth length ($r = 0.30$ for 58 boys and girls) there was no logical reason to connect the father's stature with the infant's fatness.

As shown in table 2, maternal weight at 3 months, the maternal 6-month weight gain, the mother's fat-free mass and gestation length were all positively and significantly related to birth weight. The sibling correlation was similarly significant and of the order of magnitude commonly encountered. Big women, with a large fat-free mass who carried their babies longer, and who gained more weight during pregnancy did have bigger babies. But maternal size, the length of gestation, and most other variables that were significantly correlated with birth weight were consistently unrelated to fat thickness at birth.

TABLE 2

Determinants of birth-fat weight and birth fat [1]

CORRELATION	SEX	N	r	Se_r
1. Maternal weight (3 mo)				
and birth weight	MF	95	0.23 [2]	0.10
... birth fat	MF	90	0.11	0.10
2. Maternal weight gain				
and birth weight	MF	95	0.21 [3]	0.10
... birth fat	MF	90	—0.04	0.11
3. Maternal fat-free weight [3]				
and birth weight	MF	71	0.34 [3]	0.11
... birth fat	MF	49	0.32 [3]	0.13
4. Gestation length				
and birth weight	MF	133	0.37 [3]	0.08
... birth fat	MF	61	0.12	0.13
5. Sibling correlation				
... birth weight	MF	61	0.28 [3]	0.12
... birth fat	MF	61	0.03	0.11

[1] Based on sex-specific T-scores, pooled for maximum sample size.

[2] Since the correlation between the maternal fat-free weight and the maternal weight of fat is —0.13, the correlations given here are substantially the same as the partial correlations.

[3] Significantly different from zero at $p = 0.05$ or better.

These findings, while disappointing insofar as birth fat was concerned, tended to confirm previous evidence that it is fat-free weight or lean body mass that is primarily involved in the body-size birth-size relationships. In fact, the highest correlation (0.49 for 50 cases) was obtained between the estimated maternal fat-free weight and the estimated fat-free weight of the newborn. The parental contribution, whether operating through the fetus or through the placental barrier seems to involve the fat-free size, while variations in the extent of fatness at birth still remain to be elucidated (*vide infra*).

3. *Fat and size in the newborn.*

Next, the relationship between fat at birth and various bodily dimensions was investigated, using anthropometric data collected at or shortly after birth, and measurements of long-bone and axial length obtained from the "birth" x-rays, as described in the previous section.

As shown in table 3, fat thickness and birth weight were moderately co-variant with a combined-sex correlation of 0.63 for the total series of 146. Expectably, the crown-heel length was also significantly and positively correlated with fat thickness at birth. Clearly, fat babies weighed more and were longer though part of the fat-weight correlation was due to the mutual relationship between size and weight.

TABLE 3

Relationship between fat thickness and body size at birth

CORRELATION BETWEEN FAT-PLUS-SKIN SHADOW AT 11TH RIB AND:	MALES			FEMALES			COMBINED [1]		
	N	r	Se_r	N	r	Se_r	N	r	Se_r
Birth weight	79	0.64	0.07	67	0.63	0.07	146	0.63	0.05
Crown-heel length	75	0.23	0.11	67	0.27	0.11	142	0.25	0.08
Spine length (T_1-L_5)	79	0.38	0.10	67	0.32	0.11	146	0.35	0.07
Humeral length	76	0.38	0.10	65	0.11*	0.12	141	0.25	0.07
Femoral length	69	0.30	0.11	59	0.13*	0.13	128	0.22	0.08

[1] Using sex-specific T-scores.

* Not significantly different from zero at $p = 0.05$ or better. Remaining 13 correlations significant.

The roentgenogrammetric measurements of skeletal size similarly indicated a positive relationship with fat thickness at birth. Spine length (T_1-L_5), humeral length and femoral length all yielded statistically significant correlations of the order of 0.2 to 0.3. Evidently, increased size associated with greater fat at birth involves both the axial skeleton and the long bones (despite the disproportionate prenatal growth of the trunk), and arm and leg length to a comparable degree, despite the relatively large size of the upper extremities at the time of birth.

These size-fat correlations further provided some indication that the technically difficult crown-heel measurement, is still as reliable an indication of length, as the more exact measurements of bone length, or rather the extent of ossification detectable on normally-exposed radiographs.

4. *Birth fat and subsequent growth.*

In turn, fat-thickness at birth was investigated in relation to subsequent size and fat, and to the rate of growth during the first three months of postnatal life. The variables thus considered included (a) leg fat at one month, as measured on antero-posterior x-rays of the left leg (for details see Garn, '56a), (b) crown-heel length and gross weight at 3 months and (c) the weight gain from birth to 3 months. Interpolation of values was not needed since the tolerance limits for the regular 3-months visit were plus or minus 1 day (*cf.* Garn and Shamir, '58, chapter 4).

As shown in table 4, the correlation between lower thoracic fat at birth and leg fat at 1 month was moderately high, 0.53 for 102 infants, thus suggesting an initially high correlation between the two areas. Expectably, both crown-heel length at three months and weight were positively correlated with birth fat, since the infants fatter at birth, started life both heavier and longer. Evidently, the developmental advantage associated with greater fatness at birth persisted despite the vicissitudes of the extrauterine environment.

TABLE 4

Fat thickness at birth and subsequent size [1]

CORRELATION BETWEEN FAT THICKNESS AT BIRTH AND:	No.	SEX	r	Se_r
1. Leg fat at 1 mo. (Garn *et al.*, '56a)	102	M&F	0.53 [2]	0.07
2. Weight at 3 months	94	M&F	0.29 [2]	0.10
3. Length at 3 months	119	M&F	0.21 [2]	0.09
4. Weight gain (birth to 3 mo.)	119	M&F	—0.27 [2]	0.09

[1] Based on sex-specific normalized T-scores.

[2] Significant at $p = 0.05$ or better.

Birth fat, however, was *negatively correlated* with weight gain during the first three months. This is shown by quartile sorts for fat as well as by the correlation coefficient. The infants in the lowest quartile for birth fat (Q_1) gained the most (2.68 kg), those in the second quartile were next (2.45 kg), infants in the third fat quartile gained 2.48 kg, while the fattest (Q_4) infants gained but 2.3 kg.

In short, babies who are fat at birth remain relatively fat during the first few postnatal months, they remain long, and (to a lesser degree) they remain heavy. They gain less weight after being exposed to the extrauterine environment, however, than do lean infants. A similar phenomenon has been noted at the 9-month level (*cf.* Garn, '56a), and in both cases the explanation appears to be the same: fatter infants being relatively advanced developmentally have shifted to the next later and slower rate of growth, while the lean infants are still engaged in the previous and more rapid rate of gain.[4]

5. *Fat and weight in diabetic progeny.*

Finally, fat thickness and weight were investigated in a series of nine diabetic progeny, all of them the offspring of known diabetic mothers, and most of them delivered by Caesarian section well before term. Initial weights ranged from 2235 gm (below the accepted limit for "prematures") to a sizable 5085 gm. The median birth weight was 3387 gm thus approximating the mean for the normal series. Antero-posterior torso x-rays, on which the fat measurements were made, were taken generally on the second day after birth, thus corresponding to the radiographs used in the normative series.[5]

TABLE 5

Birth-weight and fat-thickness in nine diabetic progeny

HOSPITAL NO.	SEX	GESTATION MENSTRUAL (days)	BIRTH WEIGHT (gm)	LT FAT THICKNESS (mm)	FAT T-SCORE[2]
107324	m	238	2,235	3.2	52
115434[1]	m	224	2,736	4.9	75
117515	m	245	3,005	3.4	55
114332	f	252	3,232	3.3	53
114189	f	259	3,388	4.1	64
108659	m	245	3,459	5.1	80+
121295	m	259	3,487	3.8	60
114994	m	266	3,770	4.3	68
116877[1]	m	term	5,085	7.1	85++
Median			3,387	4.4	70

[1] Vaginal delivery, all other Caesarian sections.

[2] From Fels combined-sex normalized T-scores.

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[4] Other studies have shown that infants who are small at birth grow more rapidly thereafter.

[5] These nine diabetic progeny, whose x-rays and protocols were furnished by Dr. Thomas Shaffer, University Hospital, Columbus, Ohio, included 7 born by Caesarian section, and 2 vaginal deliveries. Gestation lengths given in table 5 refer to menstrual days.

Despite the shortened length of gestation, the diabetic progeny were not limited in subcutaneous fat thickness. In fact, with fat thicknesses of 3.2 mm to 7.1 mm, none of the diabetic progeny fell *below* the median value for the normative series (see table 5). The "leanest" of the diabetic progeny had—at 32 developmental weeks of gestation and a birth weight of 2325 gm—a fat thickness equivalent to that of a full-term infant weighing nearly 1100 gm more! Several of the nine diabetic progeny exceeded the maximum fat values for the normative series of 146. Comparing the diabetic group to the normative series, using a modified sign test, the value of Chi-squared after correction for continuity was 7.1. The T-scored values similarly indicated a highly signicant fat difference between the diabetic progeny and the normal neonates.[6]

Plotting the actual fat and weight values for the diabetic series against the scattergram for the Fels newborn, the fat-to-weight relationship was even more dramatic. The diabetic progeny apparently followed the usual tendency for increased weight to parallel increased subcutaneous fat, as shown in figure 3, but suggested a greater value for the intercept *b*. In fact, there was a tendency for the two groups to separate completely, the bulk of the diabetic progeny falling above the 85th percentile for fat. With a fat/weight ratio of 1.3 as compared with 0.88 for normal neonates, it was evident that the offspring of known diabetic mothers were excessively fat for their weight, approximately 40% fatter than the progeny of putatively normal women.

DISCUSSION

The findings in this study provide a definite indication that the thickness of the subcutaneous fat can be measured reliably and profitably in the newborn, fortunately at a site with a built-in landmark that may be followed throughout life. These data, based on clinically-normal developmentally-mature white singlings, attest to the fact that fat is an extraordinarily variable "body compartment" even at birth. They further confirm the growing evidence that the female is relatively fatter than the male at all developmental horizons so far investigated. The tendency for the female neonate to gain weight less rapidly than the male neonate which is true for white and Negro infants alike (*cf.* Kasius *et al.*, '57) may in part be explained by her greater developmental maturity and relatively more ample fat reserves at birth.

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[6] Since serial antero-posterior chest x-rays were not available for the nine diabetic progeny, it is impossible to rule out the chance that the measured thickness of LT fat was to some extent exaggerated by fluid accumulation. However, excellent differentiation between the fat-plus-skin shadow and the remaining soft tissue made it less likely that the measurements were highly affected by oedema.

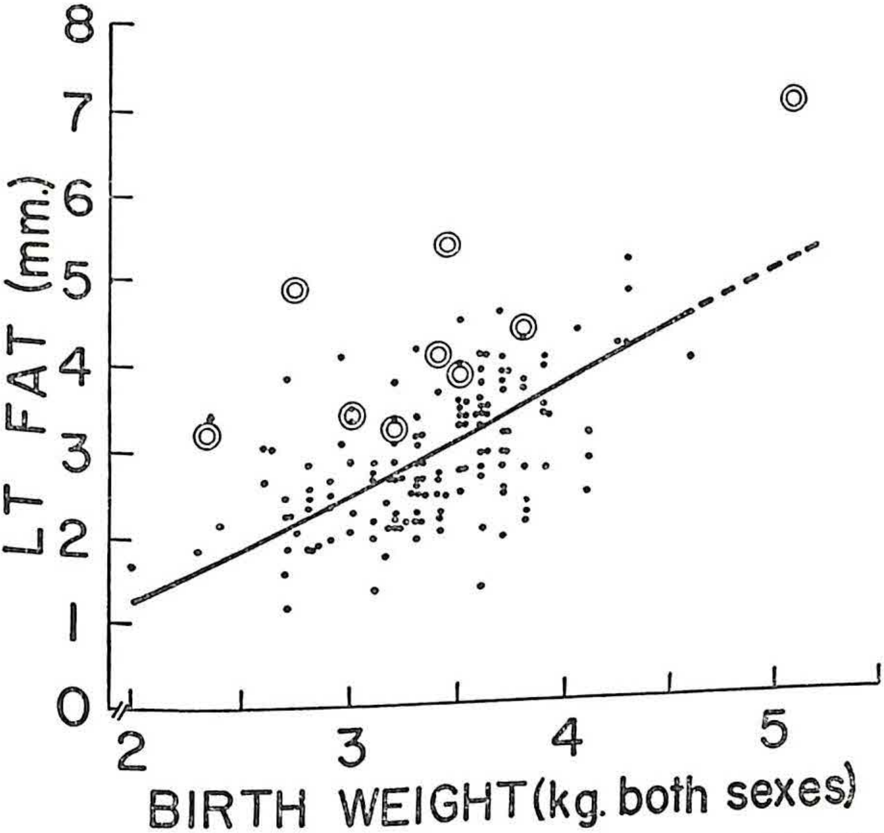

FIG. 3. NINE DIABETIC PROGENY (DOUBLE CIRCLES) PLOTTED ON THE COMBINED-SEX FAT-WEIGHT SCATTERGRAM FOR 146 FELS NEONATES. THOUGH THE MEDIAN WEIGHTS FOR THE NORMAL AND DIABETIC PROGENY WERE ALMOST IDENTICAL, THE OFFSPRING OF DIABETIC MOTHERS WERE NOTABLY FATTER FOR THEIR WEIGHT.

Within the normal variables considered in the present investigation, we have not been able to find evidence as to why one infant is fatter than another at the time of birth. Parental size, especially the fat-free mass of the mother, relates to both size and weight in the newborn, but neither maternal size nor weight gain during pregnancy significantly bear on differences in subcutaneous fat. Naturally, these findings do not contradict the evidence that *extreme* caloric deprivation does limit birth size (*cf.* Antonov, '47; Smith, '47), nor do they deny that infants carried to term and beyond may be fatter than those prematurely born. Rather, these negative findings indicate the need for a further investigation of the very suggestive evidence that the degree of fetal activity during later pregnancy may affect the extent of fat stored by the fetus *in utero* (Sontag, '40).

Clearly, fat babies are bigger babies; they weigh more and they are larger. Both upper and lower limbs of fat neonates tend to be longer, and this trend toward great length is shared by the axial skeleton as well. Yet the correlations are low; there are fat small infants, and lean large infants. As is true during the whole period of postnatal growth and and development, the fat-size relationship while positive does not give the impression that calories stored in the form of fat are readily mobilized for increased growth and a markedly augmented rate of development. In other words, one wonders why calories are stored in the form of fat, at a time when they could be most profitably used to speed ossification, protein-synthesis and general growth.

Inclusion of the diabetic progeny does bring in one variable that bears on relative fatness. Though carried to term in only one instance, all nine of the offspring of known diabetic mothers were above the norm for fat; all were exceptionally fat in relation to their weight. While an increase in available sugar is a likely explanation for the increased fat storage in the diabetic progeny, the complexity of fat metabolism and the varying endocrine abnormalities grouped under the diagnostic entity of "diabetes" indicate the need for caution in accepting the sugar hypothesis. At the same time, it may well be that fat infants are an indication of a diabetic or pre-diabetic state on the part of the mother, a possibility that can be tested by the glucose-tolerance test.

SUMMARY

1. The thickness of the fat-plus-skin shadow at the level of the 11th rib was measured on antero-posterior "birth" x-rays of 146 Ohio-born white infants, and investigated in relation to maternal size, gestation length, body size and long-bone length.

2. As with other fat sites analyzed to date, the distribution of LT fat was markedly skewed. While there was no sex difference in the

thickness of fat, the fat/weight ratio for female neonates was significantly higher than in the male.

3. Unlike birth size and birth weight which were positively and significantly correlated with maternal size, the rate of maternal weight gain, etc., fat thickness at birth was not significantly related to maternal size, weight gain or the length of gestation.

4. Fat thickness was moderately related to birth weight in both sexes ($r = 0.6$ to 0.7), and length, femoral and tibial length and spine length (T_1-L_5) to a lesser degree ($r = 0.2$ to 0.4). Fatter babies were longer overall, and in consequence of increased fatness and greater length tended to weigh more.

5. Those neonates who were fatter at birth, tended to be fatter and longer during the first few months of postnatal life, but gained less weight during early extrauterine existence.

6. Analysis of fat-weight data on a series of nine diabetic progeny revealed that the diabetic infants were extremely fat, exceptionally so in relation to their weight, despite the fact that they had been delivered well before term.

7. These findings indicated that subcutaneous fat could be invesgated roentgenogrammetrically at the earliest age levels, and with anticipation of useful findings on the fat "compartment" and its implications in terms of late prenatal development.

8. Attention was drawn to available sugar as one possible determinant of birth fat, and to variations in fetal activity as a second variable possibly affecting fat storage in the late prenatal period.

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HAND CLASPING IN DIFFERENT ETHNIC GROUPS *

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INTRODUCTION

GENETIC traits with high but variable frequencies in different populations are especially useful in evaluating and analyzing evolutionary forces. These traits may also have considerable anthropological interest in situations where classification of a population is desired. The search for genetic characters which are common in populations may be, then, highly rewarding.

The present paper will review the work of several authors on the familial and population aspects of hand clasping and will show, with data obtained in Brazil, how this trait can be used in population studies.

REVIEW OF THE LITERATURE

When an individual clasps his hands one of two mutually exclusive events occurs: either the right (situation *R*; see fig. 1 in Rothschild, '30) or the left thumb (situation *L*; see fig. 2 in Winchester, '51) emerges uppermost.

The first person to study this trait was Lutz ('08), in a random sample of families obtained in Scotland. His data suggested the presence of a genetic component, but no simple genetic mechanism fit the data. No sexual bimodality in the trait, nor reproductive selection, nor assortative mating, nor relation to handedness was found. Early manifestation of the trait was noted, and the thumb position was quite constant even in very young children. *R* individuals were found to be in the majority in the parental (59%) as well as in the filial generation (60%).

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The second paper to deal with this subject (Downey, '26) was based on American data, and was more concerned with the relation between the trait and handedness. In a non-random sample (including a high proportion of ambidextral and left-handed individuals), Downey confirmed that *R* persons are the majority (51%) but failed to support Lutz's conclusion regarding the independence of hand clasping and handedness.

Dahlberg ('26) determined the incidence of the trait in a series of 69 pairs of monozygotic and 123 pairs of dizygotic twins, and found no evidence of genetic factors. The examination of 854 school children revealed a frequency of 49.9% *R*. On the basis of these two series of data, Dahlberg concluded that "there is hardly reason to presume heredity."

Wiener ('32) studied a series of American families and confirmed the finding that *R* persons are more common (54%), and that the trait is not associated with sex or handedness. His data failed to support the hypothesis of heredity proposed by Lutz to account for the heterogeneity among the children of the different types of matings, thus confirming Dahlberg's conclusion.

Data obtained in Japan and Korea by Yamaura ('40) strongly supported the genetic hypothesis, and again *R* individuals were the more common (55% in both samples). The different frequencies detected in the sexes were ascribed to errors of sampling.

The extensive material collected in Japan by Kawabe ('49) support the notion that the trait is heritable. No clear mode of inheritance, however, could be deduced from the data. *R* individuals were the more common in all his sub-samples (with an average frequency of 64%), and no statistically significant differences were found between males and females. Kawabe's data suggest that there is some degree of association between the trait and other manual actions, and that the incidence of *R* is higher among people more than 20 years old (62% against 57%). This finding confirmed the report of Rothschild ('30), who found 48% *R* among adults and 43% *R* among children in Germany. Rothschild's and Dahlberg's data are the only data in the literature which show frequencies of *R* below 50%. Kamm ('30) and Ludwig ('32) found 50% *R* in Germany.

TABLE 1

Frequencies of R individuals among the children of the three types of matings, with statistical analysis of the distributions and calculations of the frequency (q) of the allele responsible for the R trait on the basis of Trankell's ('55) model.

Data I (original), II (from Lutz, '08), III (from Wiener, '32), IV (from Yamaura, '40), and V (from Kawabe, '55).

	CHILDREN				
	R				
PARENTS [‡]	*n*	%	TOTAL	*q*	x^2 [†]
I. $R \times R$	156	69.96	223	.9448 [*]	*1.* 11.95 ($P <.01$) ; *2.* 6.60 ($P <.02$) ; *3.* 5.50 ($P <.02$) ; *4.* 9.37 ($P <.01$) ; *5.* 2.93 ($P >.05$) ; *6.* 1.59 ($P >.20$) ; *7.* 10.28 ($P <.01$).
$R \times L$	108	57.75	187	.9455 [*]	
$L \times L$	27	48.21	56	.9473 [*]	
II. $R \times R$	166	72.49	229	.9100	*1.* 30.58 ($P <.01$) ; *2.* 13.45 ($P <.01$) ; *3.* 17.67 ($P <.01$) ; *4.* 28.98 ($P <.01$) ; *5.* 2.34 ($P >.10$) ; *6.* 6.33 ($P <.02$) ; *7.* 23.99 ($P <.01$).
$R \times L$	147	56.54	260	.9104	
$L \times L$	46	42.20	109	.8996	
III. $R \times R$	76	53.52	142	.9960	
$R \times L$	131	55.98	234	$\sqrt{-649.74}$	*1.* 3.15 ($P >.20$)
$L \times L$	42	45.16	93	.9407	
IV. $R \times R$	112	70.44	159	.8895	*1.* 30.34 ($P <.01$) ; *2.* 12.66 ($P <.01$) ; *3.* 18.02 ($P <.01$) ; *4.* 28.77 ($P <.01$) ; *5.* 2.15 ($P >.10$) ; *6.* 8.32 ($P <.01$) ; *7.* 21.93 ($P <.01$).
$R \times L$	110	52.13	211	.9156	
$L \times L$	21	31.82	66	.8643	
V. $R \times R$	594	77.75	761	.9101	*1.* 176.15; *2.* 56.81; *3.* 124.56; 4. 169.38; 5. 14.62; *6.* 40.49; *7.* 131.90; ($P <.01$).
$R \times L$	244	57.28	426	.9192	
$L \times L$	62	30.24	205	.8698	

[*] Assuming *L* to be due to a recessive gene, the gene frequencies for each group of parents, according to Trankell's model, would be .8515, .7602, and .8130 respectively. The probability that the first value would fit all the family data is less than .01; for the second, $P <.05$; for the third, $P >.20$. The hypothesis cannot be assumed to explain the whole family information.

[†] *1* refers to x^2 for $(R \times R)\,\mathrm{vs}\,(R \times L)\,\mathrm{vs}\,(L \times L)$; *2* to $(R \times R)\,\mathrm{vs}\,(R \times L)$; *3* to $(R \times R) + (R \times L)\,\mathrm{vs}\,(L \times L)$; *4* to $(R \times R)\,\mathrm{vs}\,(L \times L)$; *5* to $(R \times R) + (L \times L)\,\mathrm{vs}\,(R \times L)$; *6* to $(R \times L)\,\mathrm{vs}\,(L \times L)$; *7* to $(R \times L) + (L \times L)\,\mathrm{vs}\,(R \times R)$.

[‡] The three classes of families in each set of data will be referred as a, b, and c respectively in the next Table.

ORIGINAL DATA AND DISCUSSION

Family data

The first section of table 1 presents the frequency of R children in a Brazilian sample of the three types of matings (parents $R \times R$, $R \times L$, and $L \times L$). The class $R \times L$ includes data from ♀ $R \times$ ♂ L and ♀ $L \times$ ♂ R; no statistically significant differences were found between these matings (56 R and 44 L among the children of ♀ $R \times$ ♂ L, and 52 R and 35 L among the children of ♀ $L \times$ ♂ R; $X^2 = .27$, $DF = 1$, $P > .50$). The latter observation holds true for the data of Lutz ('08), ($X^2 = .09$, $DF = 1$, $P > .70$), Wiener, 1932 ($X^2 = .03$, $DF = 1$, $P > .80$), and Kawabe, 1949 ($X^2 = .18$, $DF = 1$, $P > .50$). Yamaura ('40) does not differentiate between these two types of matings. X^2 for heterogeneity among the three types of Brazilian matings (table 1) is 11.95 ($DF = 2$, $P < .01$). Partition of this X^2 (see table 1) reveals that the children of parents $R \times L$ have frequencies of R identical to those found among the children of $R \times R$ and $L \times L$ combined (a fact also seen in the data of Lutz, '08, and Yamaura, '40, but not of Kawabe, '49). Our data also show that the differences between the children of $R \times L$ and $L \times L$ are not significant, an observation not confirmed by the above authors who found significant differences. All the other comparisons in our data show significant differences.

Wiener's data ('32) differ from those of Lutz, Kawabe, Yamaura, or our own in that no significant differences between the various mating types were found. A clear suggestion of a parent-offspring correlation in hand-clasping type is found in all data save Wiener's. In the absence of a psychological explanation of the findings, it is our opinion that some prenatal factor (probably genetic in nature) is responsible for the trait. No simple Mendelian hypothesis is capable, however, of explaining the findings.

We have applied a genetic model suggested by Trankell ('55) to our data as well as to those presented by Lutz, Wiener, Yamaura, and Kawabe (*cf.* table 1). The model is that R is due to a recessive gene (r), but that a certain fraction of rr individuals are phenotypically L; the opposite hypothesis (that L individuals are homozygous for a recessive gene (l), but some ll individuals are phenotypically R) does not fit our data (see footnote to table 1). Three of the 5 sets of data (Lutz's, Yamaura's, and our own) give results compatible with the first model, but Kawabe's data does not (*cf.* statistical analysis in table 2).

Wiener's data lead to an absurd result (table 1). No conclusions can be drawn from the analysis based on Trankell's model.

The frequencies (in per cent) of the 4 types of matings ($R \times R$, ♀ $R \times$ ♂ L, ♀ $L \times$ ♂ R, and $L \times L$) in our data are, respectively, 47.80,

TABLE 2

Statistical analysis of some values of q in each set of data presented in Table 1, according to Trankell ('55).

	Family *a* † Children		Family *b* † Children		Family *c* † Children			
q	*R*	*L*	*R*	*L*	*R*	*L*	x^2	*P*
I * →	156	67	108	79	27	29		
.94	157.6	65.4	106.9	80.1	25.9	30.1	.1687	.50-.70
.95	154.3	68.7	108.8	78.2	27.4	28.6	.0863	.70-.80
II →	166	63	147	113	46	63		
.90	169.7	59.3	145.5	114.5	46.1	62.9	.3470	.50-.70
.91	166.0	63.0	147.0	113.0	48.0	61.0	.1489	.70
IV →	112	47	110	101	21	45		
.86	119.8	39.2	102.1	108.9	20.5	45.5	3.2619	.05-.10
.89	111.9	47.1	106.7	104.3	24.0	42.0	.7961	.30-.50
.91	107.0	52.0	109.3	101.7	26.3	39.7	2.4993	.10-.20
.92	104.7	54.3	110.5	100.5	27.5	38.5	4.1289	.02-.05
V →	594	167	244	182	62	143		
.87	650.1	110.9	216.7	209.3	62.1	142.9	40.2203	<.01
.91	594.2	166.8	239.5	186.5	83.0	122.0	9.1214	<.01
.92	581.3	179.7	244.4	181.6	88.3	116.7	14.9370	<.01

* Numbers I, II, IV and V as in table 1.

† *a*, *b* and *c* correspond respectively to the three classes of families described in table 1.

22.64, 18.87, and 10.69 ($N = 159$). These frequencies do not differ significantly from those expected on the basis of random mating ($X^2 = .24$; $DF = 3$, $P > .95$). The 4 classes of parents have had the following average numbers of children: 2.93 ± .21, 2.78 ± .29, 2.90 ± .38, and 3.29 ± .50, respectively. These differences are not statistically significant.

The frequencies of *R* children by birth order in the pooled data are

60% among first born children ($N=62$), 45% among second born ($N=51$), 63% among third born ($N=30$), and 61% among fourth born and the following children ($N=33$). There is no significant birth rank effect ($X^2=3.74$, $DF=3$, $P>.20$).

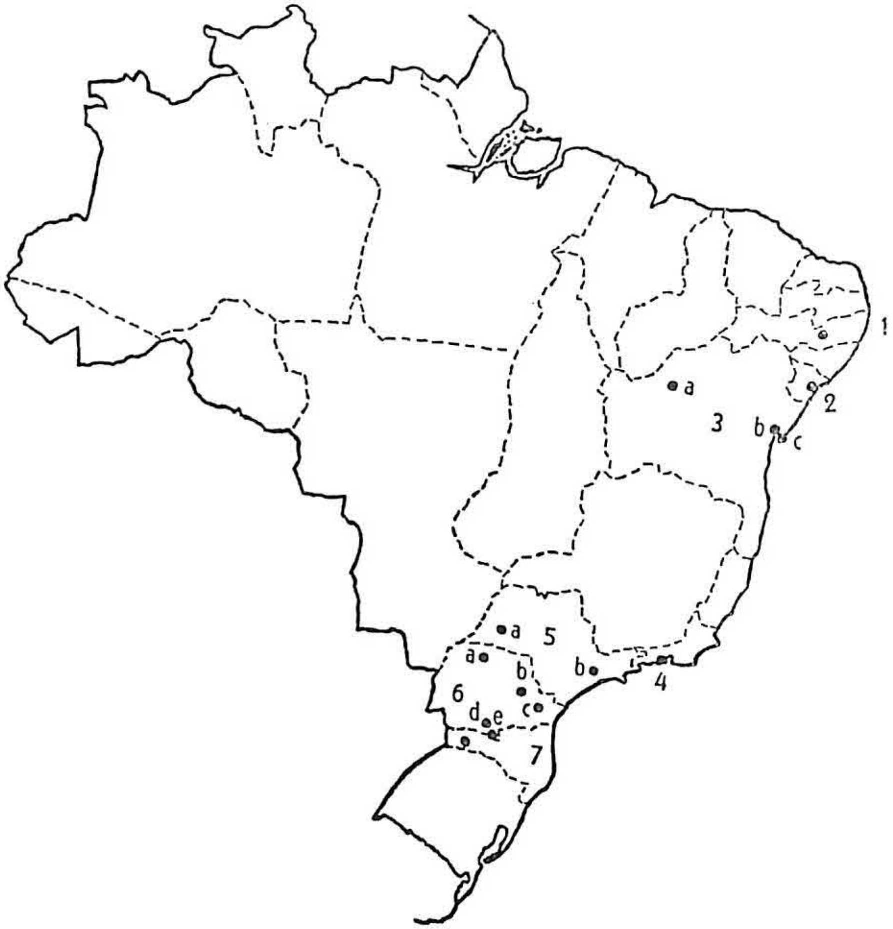

FIG. 1. STATES (IN ITALIC) AND LOCALITIES WHERE THE DATA HAVE BEEN COLLECTED. 1. *Pernambuco*, PESQUEIRA; 2. *Sergipe*, ARACAJU; 3. *Bahia*, a. BARRA DO RIO GRANDE, b. SANTO AMARO, c. SALVADOR; 4. *Federal District*, RIO DE JANEIRO; 5. *São Paulo*, a. PRESIDENTE PRUDENTE, b. SÃO PAULO; 6. *Paraná*, a. LONDRINA, b. CASTRO, c. CURITIBA, d. MANGUEIRINHA, e. PALMAS; 7. *Santa Catarina*, XAPECÓ.

Population data

Table 3 presents a series of samples drawn from several different Brazilian populations (see also fig. 1). These samples consist of

TABLE 3

Frequencies of R individuals in different localities and ethnic groups in Brazil

LOCALITIES AND ETHNIC GROUPS	SAMPLE SIZE	INDIVIDUALS *R*	
		n	%
Rio de Janeiro, DF (C, S)			
Caucasians	827	454	54.90
Mulattoes	531	317	59.70
Negroes	98	63	64.29
Total	1,456	834	57.28
São Paulo, SP (C, S)			
Caucasians	582	320	54.98
Mulattoes	107	62	57.94
Negroes	36	28	77.78
Total	725	410	56.55
Mongoloids (J, C)	375	215	57.33
Salvador, Ba (C, Ne, S)			
Caucasians	157	90	57.32
Mulattoes	439	283	64.46
Negroes	355	245	69.01
Total	951	618	64.98
Londrina, Pr (C, S)			
General sample (W)	237	142	59.92
Mongoloids (J)	637	398	62.48
Palmas and Xapecó, Pr (A)			
Caingang Indians	167	88	52.69
Mangueirinha, Pr (A)			
Guarani Indians	25	17	68.00
Curitiba, Pr (C, W)	1,502	851	56.66
Castro, Pr (C, W)	308	187	60.71
Presidente Prudente, SP*, (A, H)	146	90	61.64
Barra do Rio Grande, Ba (C,ne)	220	148	67.27
Santo Amaro, Ba (C, Ne)	91	66	72.53
Aracaju, Se (C, W)	146	76	52.05
Pesqueira, Pe (C, W)	476	259	54.41
Total of Caucasians	1,566	864	55.17
Total of Mulattoes	1,077	662	61.47
Total of Negroes	489	336	68.71
Total of Indians	192	105	54.69
Total of Mongoloids	1,012	613	60.57
GRAND TOTAL	7,462	4,399	58.95

A—mainly adults; C—mainly or exclusively school children; H—highly heterogeneous sample from the ethnic view-point; J—Japanese stock; Ne—mainly Negroes and Mulattoes (frequencies higher than 80%); ne—43% Negroes and Mulattoes; W—mainly Caucasians; * including also a few data obtained in neighboring localities; S—samples not representative of the whole population, since they have been obtained in wards showing high frequencies of Negroes and/or Mongolians.

unrelated individuals. The main conclusions to be drawn from these data are the following:

I. *R* individuals are the more frequent in all samples. This has been previously reported by Aguiar and Freire-Maia ('53).

II. Analysis of the total Negro, Mulatto, and Caucasian samples reveals significant heterogeneity ($X^2 = 31.11$, $DF = 2$, $P < .01$). Similar analysis of the subsamples from Salvador, São Paulo, and Rio de Janeiro yield X^2 equal to 6.63 ($P < .05$), 7.27 ($P < .05$), and 5.15 ($P > .05$), respectively. Negroes show frequencies of *R* (68.71% in the total) significantly higher ($X^2 = 28.11$, $DF = 1$, $P < .01$) than Caucasians (55.17% in the total). The Mulatto subsamples exhibit intermediate frequencies, as expected (61.47% in the total).

III. The differences between 11 localities and two racial groups given in table 3 (obtained by using the total from each locality and pooling the data on Mongolians and Indians into two groups irrespective of the locality from whence they come) are highly significant ($X^2 = 44.59$, $DF = 12$, $P < .01$). This X^2 value is, however, largely due to the three samples from the state of Bahia (Salvador, Santo Amaro, and Barra do Rio Grande); their contribution to the total X^2 is 27.53. This is probably due to the high frequencies of Negroes and Mulattoes in these areas (70% in Bahia according to the 1950 Census). Upon removal of these three samples, the remaining samples are homogeneous ($X^2 = 11.78$, $DF = 9$, $P > .20$).

IV. The differences between the three Caucasian groups ($X^2 = .33$, $DF = 2$, $P > .80$), between the three Mulatto groups ($X^2 = 2.93$, $DF = 2$, $P > .20$), between the three Negro groups ($X^2 = 2.28$, $DF = 2$, $P > .30$), between the two Indian groups ($X^2 = 2.06$, $DF = 1$, $P > .10$), and between the two Mongolian subsamples ($X^2 = 2.62$, $DF = 1$, $P > .20$) are not significant. The subsamples within each ethnic group have been pooled to form larger samples for intergroup comparisons. The differences between Indians and Caucasians ($X^2 = 0.02$, $DF = 1$, $P > .80$), Indians and Mulattoes ($X^2 = 3.13$, $DF = 1$, $P > .05$), Indians and Mongolians ($X^2 = 2.32$, $DF = 1$, $P > .10$) are not significant, but that between Indians and Negroes is highly significant ($X^2 = 11.88$, $DF = 1$, $P < .01$). The differences between Mongolians and Caucasians ($X^2 = 7.33$, $DF = 1$, $P < .01$) and between Mongolians and Negroes ($X^2 = 9.39$, $DF = 1$, $P < .01$) are highly significant, but that between Mongolians and Mulattoes ($X^2 = .18$, $DF = 1$, $P > .50$) is not significant.

V. One sample of 1,143 children and teen-age boys and girls (53% *R*) obtained in elementary and high schools in two localities, Curitiba and Castro, in the state of Paraná, is significantly different ($X^2 = 27.84$, $DF = 1$, $P < .01$) from a sample of 403 adults from the same areas (68% *R*). Analysis of these data, taking sex into account, yields similar results. Thus, among 351 females, 60% of 186 children and 70% of 165 adults were *R* ($X^2 = 3.91$, $DF = 1$, $P < .05$), and among 1,138 males, 52% of 900 children and 67% of 238 adults were *R* ($X^2 = 17.17$, $DF = 1$, $P < .01$). These findings with respect to sex are in agreement with those of Kawabe ('49), who detected a significant difference among individuals above and below the age of 20 years (see also Rothschild, '30).

The Caucasian fraction of the non-adult sample obtained in Curitiba (which forms a large part of the above-mentioned sample from the State of Paraná) has been subdivided according to age into three groups (8–12, 13–16, and 16–20). The frequencies of *R* individuals in these groups were 54%, 53%, and 48% respectively. These differences are not significant ($X^2 = 2.22$, $DF = 2$, $P > .30$). No significant difference has been found in a sample of 926 children from Salvador, Ba, classified into three groups according to age (7–9, 10–12, and 13–16) with 68%, 63%, and 66% *R* respectively ($X^2 = 2.12$, $DF = 2$, $P > .30$). The analysis within each of the three ethnic components of the whole Salvador sample also failed to reveal an age effect.

VI. An analysis of the incidence of *R* individuals among males (56%) and females (61%) shows a highly significant difference in a total of 4,882 individuals including Caucasians, Mulattoes, and Negroes ($X^2 = 15.95$, $DF = 1$, $P < .01$). Within each of the ethnic subsamples, the sex differences are significant only among Caucasians (53% in males and 57% in females; $N = 3{,}040$; $X^2 = 5.61$, $DF = 1$, $P < .02$). Among Negroes and Mulattoes the same excess of *R* among females occurs, however (73% versus 65% in Negroes, $N = 489$; and 64% versus 60% in 1,353 Mulattoes). Among 190 Indians, 948 Mongolians, and 403 adults mostly Caucasians from Paraná, the excess of *R* females also proves to be not significant. A significant sex difference does occur in a predominantly Caucasian sample of 1,086 children from Paraná (52% in males and 60% in females; $X^2 = 4.41$, $DF = 1$, $P < .05$). No sex effect has been found by the other authors who have studied this trait, but the consistency of the excess of *R* females among all of the comparisons in our data leads us to believe that the sexes do differ in the frequencies of this trait.

VII. Since the frequencies of the two forms of the trait in different samples are influenced not only by the ethnic composition of the sample but probably also by the age and sex distribution of the individuals involved, it is hazardous to compare samples obtained by different authors unless the above characteristics are clearly specified. These characteristics are not sufficiently specified in the data available in the literature to permit more than a rough comparison of our data with those in the literature. Our Caucasian samples, for instance, with 55% *R* are intermediate between those from Germany and Sweden (50% and less) and those from Scotland (parents in Lutz's family data, 60%), and are identical with the American data reported by Wiener. Our Mongolian samples, with 61% *R*, are roughly comparable to the three Japanese samples of Kawabe (60%, 64%, and 65%) but are unlike Yamaura's sample from Japan and Korea (55%).

SUMMARY

Family data show some parental influence on the type of hand clasping encountered among their children. A prenatal factor (probably genetic in nature) is suggested as the explanation of this correlation. No simple Mendelian mechanism seems to account for the data, however. The four types of matings occur with the frequencies expected from random mating, and the four mating types appear equi-fertile. Birth order exerts no effect on the type of hand clasping.

Population data show that Negroes and Caucasians, Caucasians and Mongolians, and Mongolians and Negroes differ in the frequencies of the two types of hand clasping. Among Mulattoes, the frequencies are intermediate between Caucasians and Negroes, as expected. No other significant difference has been detected among the various ethnic samples. Adults have a higher frequency of the "right thumb" type than children, and females a higher frequency than males.

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HEIGHT AND WEIGHT OF SELECTIVE SERVICE REGISTRANTS PROCESSED FOR MILITARY SERVICE DURING WORLD WAR II

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INTRODUCTION

THE height and weight findings presented here relate to Selective Service registrants examined for military service during the thirteen-month period from January 1943 through January 1944. Somewhat over 5.5 million men, 18 through 37 years of age, were examined during this period by the Joint Army and Navy Induction Stations.

Each registrant arriving at an induction station was ordinarily given a complete medical examination in order to determine his acceptability for military service. The results of the examination were recorded on DSS Form 221 ("Report of Physical Examination and Induction") which, in addition to the clinical evaluation of the registrant's medical (physical and psychiatric) status, included related laboratory (x-ray findings, serologic tests for syphilis, etc.) and anthropometric data (height, weight).

The induction stations were required to submit a copy of these physical examination forms to the office of The Surgeon General, Army, for: *a.* Each examined registrant who was disqualified for military service, and *b.* each qualified examinee assigned to the Army. During this period about three-fourths of the qualified registrants called for induction were assigned to the Army and about one-fourth to the Navy. Copies of the physical examination forms for Navy inductees were not available to The Surgeon General's Office, Army. Adjustments, explained later, were made for this partial coverage of the inducted registrants, in order to reflect the total examined registrant population.

Out of the total number of the physical examination forms received during this period a ten-percent random sample was drawn by choosing a single terminal digit of the Selective Service number assigned to the registrant by his local board, as recorded on the forms. The punch-cards, to which the height-weight data obtained from the sample were transferred, contained pertinent personal items, such as the examinee's occupation, place of residence, age, race, etc., and related medical data.

About 465,000 medical examination forms were included in the study. A distribution of these forms by age, race, and military qualification (inducted or disqualified) of the examinees involved in the study is shown in table 1. Age is given as of last birthday. The term "white" used in the racial classification refers to non-Negro. The data relate to examinees of known height and weight, as well as of known age and race.

HEIGHT AND WEIGHT MEASUREMENTS

The examinees were measured without their shoes and weighed without clothing. Height was recorded in inches, and weight in pounds, as specified on the physical examination forms.

Specific instructions with respect to measuring height have been provided by the regulations on physical standards (Mobilization Regulations 1-9, '42) which read as follows:

> "Use a board at least 2 inches wide by 80 inches long, placed vertically and carefully graduated to $\frac{1}{4}$ inch between 58 inches from the floor and the top end. Obtain the height by placing vertically, in firm contact with the top of the head, against the measuring rod an accurately squared board of about 6 by 6 inches, best permanently attached to graduated board by a long cord. The individual should stand erect with back to the graduated board, eyes straight to the front."

An examination of the physical examination forms revealed that ordinarily height was recorded in whole inches, though it was given sometimes to a half or quarter inch. Recorded fractions of inches were dropped in coding the height data.

Weight was presumably taken to the nearest pound. Fractions of pounds appearing on the forms were disregarded, and the weight was coded in whole numbers, as in the case of height.

TABULATION OF DATA

In tabulating the height and weight data, the qualified (inducted) and disqualified examinees were treated separately. This was done for two reasons: First, it was important from a military point of view to obtain separate height and weight data on individuals qualified for military service; second, anticipated differences in the height and weight of these groups suggested adjustments for the under-presentation of the qualified group—due to the unavailability of the physical examination forms of Navy inductees—in combining these data for inducted and disqualified examinees. (See following section: Adjustments for Disproportionality.)

TABLE 1

Number of physical examination forms included in the sample study of height and weight, by age, race, and military qualification of the examinees (January 1943 through January 1944) *

AGE	WHITE		NEGRO	
	Inducted	Disqualified	Inducted	Disqualified
18–19	77,744	32,507	9,171	6,968
20–24	67,824	29,971	11,559	9,572
25–29	40,934	28,971	6,661	9,446
30–34	34,567	34,225	5,407	10,897
35–37	16,303	22,891	2,535	6,422
Total (18–37)	237,372	148,565	35,333	43,305

* Source: Sample tabulations of DSS Form 221 ("Report of Physical Examination and Induction").

Weight by height was cross-tabulated for each of these groups, as follows: *a.* By age and race, *i. e.*, for each subgroup, as given in table 1; *b.* by geographic region and race; *c.* by size of community and race, and *d.* by occupation and race.

ADJUSTMENTS FOR DISPROPORTIONALITY

Because of differences found between the height and weight values of the qualified (inducted) and those of the disqualified groups, direct additions of the height and weight distributions of these groups—for the purpose of obtaining corresponding distributions and derived values (means, standard deviations, etc.) for the combined (inducted plus disqualified) groups—would obviously have introduced certain bias in the calculations by giving undue weight to disqualified groups. To correct for these disproportionalities, the cross-tabulated weight-height data of the qualified (inducted) examinees were proportionally increased, before combining these with the corresponding data for the disqualified groups. These adjustments were made within the height-range of 62 through 76 inches—the acceptable height-range for Navy inductees. (For Army inductees, the acceptable height-range was from 60 through 78 inches.)

In making these adjustment, it was assumed that within this adjusted height-range, the Navy inductees were distributed by height and weight as the Army inductees, as no other factors related to height or weight played a decisive part in the assignment of qualified registrants to the Army or the Navy. (See: "Appendix: Technical Notes," par. 2: Adjustment Factors.)

REPRESENTATIVENESS OF THE DATA

Limited to registrants examined at the induction stations, these height and weight data excluded registrants disqualified by the local boards prior to the induction processing, as well as deferred and exempt registrants. During this period the local boards played a minor role in the screening of registrants for military service, so that the exclusion of registrants disqualified by the local boards could not have produced an appreciable distorting effect on the height and weight data. As to the deferred and exempt registrants, there is no reason to believe that their distributions by height and weight differed materially from that of the other registrants. There are indications that such differences would be primarily a function of age. But these differences have been essentially eliminated by the fact that the height and weight values were derived by age.

With the above-mentioned adjustments for disproportionality, the height and weight distributions and the derived height and weight values presented here may be reliably regarded as representative of all registrants, by age and race, within the (18–37) age range.

FREQUENCY DISTRIBUTIONS OF HEIGHT AND WEIGHT AND THEIR QUARTILE VALUES

As first step in the analysis of the data, two basic tables (tables 2-3) are presented. Table 2 shows distributions of the examined registrants by height, in terms of age and race; table 3 shows corresponding distributions by weight. (Similar distributions are available for inductees alone.)

TABLE 2

Percent Distribution of Selective Service registrants examined for military service by height, age, and race

(January 1943 through January 1944)

HEIGHT IN INCHES		PERCENTAGE DISTRIBUTION, BY AGE									
		WHITE					NEGRO				
As Tabulated	Mid-values	18–19	20–24	25–29	30–34	35–37	18–19	20–24	25–29	30–34	35–37
Total		100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
<60	58.9	0.08	0.07	0.10	0.15	0.21	0.13	0.11	0.11	0.14	0.22
60	60.2	0.20	0.19	0.20	0.32	0.35	0.31	0.22	0.27	0.34	0.39
61	61.2	0.47	0.41	0.48	0.62	0.72	0.77	0.61	0.64	0.75	1.00
62	62.2	1.13	1.08	1.19	1.55	1.97	1.60	1.37	1.43	1.69	1.90
63	63.2	2.62	2.30	2.39	3.04	3.63	3.00	2.84	2.89	3.56	3.92
64	64.2	4.84	4.60	4.82	5.78	6.55	6.33	5.49	5.19	6.06	6.84
65	65.2	8.36	7.87	8.38	9.23	10.14	10.11	9.24	8.99	9.74	10.21
66	66.2	12.16	11.46	11.72	12.29	12.78	13.79	12.40	12.77	12.98	13.76
67	67.2	14.72	14.59	14.78	15.05	15.51	15.40	15.00	15.03	15.22	15.12
68	68.2	15.64	15.55	15.67	15.31	14.88	14.68	15.59	15.16	15.02	13.26
69	69.2	13.96	14.28	13.86	13.25	12.46	13.07	12.88	13.45	12.43	13.27
70	70.2	10.56	11.11	10.73	9.78	8.96	9.36	9.72	9.77	9.25	8.59
71	71.2	7.28	7.83	7.48	6.65	6.04	5.76	6.86	6.90	6.17	5.92
72	72.2	4.23	4.67	4.44	3.77	3.26	3.17	4.13	4.21	3.60	3.12
73	73.2	2.12	2.27	2.11	1.77	1.40	1.52	1.97	1.66	1.69	1.29
74	74.2	0.94	1.01	0.99	0.86	0.69	0.60	0.91	0.89	0.79	0.66
75	75.2	0.43	0.41	0.38	0.33	0.26	0.25	0.32	0.36	0.31	0.30
76	76.2	0.17	0.18	0.15	0.15	0.12	0.09	0.16	0.14	0.15	0.12
77	77.2	0.05	0.07	0.07	0.05	0.03	0.02	0.10	0.07	0.06	0.07
78+	78.3	0.04	0.05	0.06	0.05	0.04	0.04	0.08	0.07	0.05	0.04
Population Bases *		135,053	119,433	82,964	79,820	44,395	17,123	22,370	16,822	16,884	9,229

* Adjusted for disproportionality. (See text.)

The distribution by height (table 2) are given in terms of one-inch intervals, as tabulated. A special study of a number of cases indicated that the heights averaged approximately two-tenths of an inch in excess of the coded (and hence tabulated) whole number of inches. (See report: Surgeon General's Office, '45.) The midvalues of the height-intervals were established accordingly. For example, the midvalue of "tabulated" 70 inches is shown in table 2 as 70.2 inches. Separate determinations were made for the midvalues of the open-end height intervals, namely, for the intervals of "under 60" and "78 and over," since these intervals were lumped. Their respective midvalues were estimated as 58.9 and 78.3 inches for all distributions.

Weight was tabulated in 5-pound intervals, and it is so presented in table 3. Since weight was supposedly recorded to the nearest pound, the midvalue, say, of the 100–104 tabulated weight-interval is shown in table 3 as 102 pounds. Examinees who weighed under 90 pounds are given in the table as one group, although their individual weights in 5-pound intervals and their frequencies were available from the tabulations. This is also true of examinees who weighed 305 pounds or more. The midvalues of these open-end weight intervals ("under 90" and "305 and over") were determined on the basis of their actual frequencies and are given in the footnotes to the weight-table (table 3).

The upper and lower limits of the height-intervals (table 2) should be taken, of course, as ∓ 0.5 inch from their midvalues (table 2), and the corresponding limits of the weight-intervals as ∓ 2.5 pounds from their midvalues (table 3).

At the bottom of each column of the tables (tables 2-3) the population base is given for the particular distribution. These population bases exceed in each case the corresponding sum of disqualified plus inducted registrants, that would be obtained from table 1, due to the corrections for disproportionality.

The height and weight distributions (tables 2-3) have been condensed in table 4 into quartile values. The following general characteristics of these values will be noted: *a.* Increase for both white and Negro examinees in the quartile values of height through the (20–24) age-interval, followed by a decline, age by age, beyond that age-interval, signifying lower heights in the advanced ages; *b.* gradual increase for white and Negro examinees in all quartile values of weight, beginning with the youngest age group, indicating larger weights with advancing age, and *c.* relatively larger quartile values of height and, generally, also of weight for white than for Negro examinees. (This is further discussed in the

TABLE 3

Percent distribution of Selective Service registrants examined for military service by weight, age, and race

(January 1943 through January 1944)

WEIGHT IN POUNDS		PERCENTAGE DISTRIBUTION, BY AGE									
		WHITE					NEGRO				
As Tabulated	Mid-values	18–19	20–24	25–29	30–34	35–37	18–19	20–24	25–29	30–34	35–37
Total		100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00	100.00
Under 90	*	0.05	0.01	0.01	0.01	0.01	0.02	—	—	0.01	—
90–94	92	0.06	0.03	0.02	0.03	0.03	0.05	0.04	0.01	0.02	0.01
95–99	97	0.24	0.11	0.09	0.10	0.15	0.16	0.09	0.05	0.06	0.12
100–104	102	0.54	0.25	0.21	0.21	0.25	0.50	0.17	0.13	0.13	0.23
105–109	107	1.34	0.72	0.55	0.53	0.59	1.10	0.46	0.36	0.36	0.44
110–114	112	2.41	1.38	1.06	1.14	1.16	2.00	1.04	0.89	0.95	1.09
115–119	117	4.14	2.64	2.02	1.95	1.94	3.88	2.03	1.66	1.93	1.68
120–124	122	6.55	4.49	3.59	3.29	3.34	6.12	3.98	3.22	2.83	2.94
125–129	127	8.64	6.51	5.15	4.75	4.41	8.83	6.17	4.90	4.76	5.08
130–134	132	10.91	9.19	7.12	6.50	6.48	11.94	8.77	7.25	7.21	6.75
135–139	137	11.11	10.00	8.15	7.35	7.34	12.04	10.45	9.21	8.74	8.05
140–144	142	11.53	10.81	9.22	8.81	8.35	12.94	12.07	10.47	9.78	9.59
145–149	147	9.69	10.14	9.17	8.77	8.22	10.89	11.77	10.73	9.91	9.68
150–154	152	8.94	10.19	9.79	9.23	8.77	10.23	12.18	12.44	11.44	10.50
155–159	157	5.93	7.29	7.61	7.32	7.38	5.97	8.22	8.43	8.00	7.92
160–164	162	4.86	6.22	6.97	7.15	7.61	4.61	6.90	7.48	7.72	7.67
165–169	167	3.38	4.71	5.74	5.99	6.05	3.11	4.93	5.87	5.98	5.93
170–174	172	2.58	3.83	5.02	5.52	5.41	1.96	3.71	4.66	4.91	4.95
175–179	177	1.73	2.68	3.93	4.39	4.46	1.23	2.21	3.52	3.51	3.93
180–184	182	1.31	2.22	3.34	3.82	4.07	0.78	1.63	2.44	2.83	3.21
185–189	187	0.93	1.52	2.49	2.77	3.07	0.46	0.94	1.60	2.16	2.51
190–194	192	0.69	1.18	2.10	2.36	2.49	0.29	0.67	1.28	1.56	1.61

195–199	197	0.52	0.93	1.50	1.77	1.97	0.20	0.38	0.77	1.24	1.31
200–204	202	0.45	0.74	1.36	1.56	1.64	0.16	0.31	0.72	1.08	1.26
205–209	207	0.31	0.49	0.86	1.13	1.19	0.08	0.24	0.42	0.60	0.87
210–214	212	0.23	0.40	0.67	0.86	0.84	0.08	0.15	0.41	0.56	0.69
215–219	217	0.19	0.29	0.49	0.63	0.68	0.07	0.08	0.24	0.41	0.37
220–224	222	0.15	0.22	0.42	0.49	0.53	0.07	0.11	0.22	0.29	0.34
225–229	227	0.11	0.18	0.32	0.33	0.35	0.02	0.06	0.13	0.28	0.24
230–234	232	0.08	0.13	0.23	0.29	0.29	0.05	0.06	0.07	0.17	0.25
235–239	237	0.07	0.11	0.16	0.20	0.20	0.01	0.06	0.12	0.11	0.23
240–244	242	0.08	0.08	0.16	0.20	0.20	0.04	0.01	0.07	0.11	0.07
245–249	247	0.06	0.05	0.11	0.10	0.12	0.02	0.04	0.04	0.08	0.08
250–254	252	0.05	0.06	0.08	0.11	0.11	0.01	0.01	0.03	0.08	0.13
255–259	257	0.03	0.04	0.05	0.09	0.07	0.02	0.03	0.07	0.02	0.05
260–264	262	0.02	0.04	0.06	0.05	0.06	0.01	—	0.01	0.04	0.03
265–269	267	0.02	0.02	0.05	0.05	0.04	0.01	0.00	0.01	0.03	0.05
270–274	272	0.01	0.02	0.03	0.03	0.03	0.01	0.01	—	0.04	0.07
275–279	277	0.01	0.01	0.03	0.03	0.02	—	—	0.01	0.02	0.03
280–284	282	0.01	0.02	0.02	0.03	0.01	0.01	0.01	0.01	—	0.03
285–289	287	0.01	0.01	0.02	0.01	0.01	—	—	0.01	—	—
290–294	292	0.01	0.01	0.01	0.01	0.02	—	0.00	0.01	0.01	—
295–299	297	0.00	0.01	—	0.00	0.01	0.01	0.01	—	0.01	0.01
300–304	302	0.01	0.01	0.01	0.01	0.02	—	—	0.01	0.01	—
305 & Over	*	0.01	0.01	0.01	0.03	0.01	0.01	—	0.02	0.01	—
Population Bases **		135,053	119,433	82,964	79,820	44,395	17,123	22,370	16,822	16,884	9,229

* *White.* *a.* For under 90, by age: (18–19)—84.5 pounds; (20–24)—85.0 pounds; (25–29)—86.0 pounds; (30–34)—83.0 pounds; (35–37)—86.0 pounds;

b. For 305 and over, by age: (18–19)—322.6 pounds; (20–24)—317.1 pounds; (25–29)—315.2 pounds; (30–34)—326.0 pounds; (35–37)—338.0 pounds

Negro. *a.* For under 90, by age: (18–19)—80.5 pounds; (30–34)—87.0 pounds; *b.* For 305 and over, by age: (18–19)—332.0 pounds; (25–29)—323.5 pounds; (30–34)—317.0 pounds.

** See footnote to table 2.

following section on the mean heights and weights and their modal values.) As might have been expected, the median height-values were found to be the same as their respective mean values due to the normality of their distributions, while the median weight-values were found to be different from their respective mean values because of their skewed distributions (table 4).

TABLE 4

Quartile values of height and weight of Selective Service registrants examined for military service, by age and race (January 1943 through January 1944)

	QUARTILE VALUES					
	White			Negro		
AGE	First	Second (Median)	Third	First	Second (Median)	Third
			HEIGHT (INCHES)			
Total: Ages (18–37)	66.2	68.0	69.7	66.0	67.7	69.5
18–19	66.3	68.0	69.8	65.9	67.6	69.4
20–24	66.4	68.2	69.9	66.1	67.9	69.6
25–29	66.3	68.1	69.8	66.1	67.9	69.6
30–34	66.1	67.8	69.6	65.9	67.7	69.5
35–37	65.8	67.6	69.4	65.7	67.5	69.3
			WEIGHT (POUNDS)			
Total: Ages (18–37)	134.3	147.3	163.1	135.5	147.3	160.3
18–19	130.0	141.2	153.9	130.5	140.8	151.7
20–24	134.3	146.4	160.5	135.6	146.5	158.0
25–29	137.7	151.4	168.2	138.0	149.9	163.0
30–34	138.9	153.1	171.2	138.4	150.9	165.5
35–37	139.0	153.9	172.2	138.6	151.6	167.2

MEAN HEIGHTS AND WEIGHTS AND MODES OF WEIGHT

Heights. The mean heights of the examined registrants are shown in table 5, by age and race. As these data indicate, the (20–24) age-group—age of maturity—had the highest stature. The younger, the (18–19) age-group—still a growing group—and all the age-groups beyond the (20–24) age-group had lower mean heights. This holds for both white and Negro examinees.

For white examinees, the mean heights were as follows: 68.06; 68.19; 68.09; 67.83, and 67.59 inches, for the (18–19), (20–24), (25–29), (30–34), and (35–37) age groups, respectively. For Negro examinees, the mean heights were 67.64; 67.91; 67.90; 67.70, and 67.51 inches, for the corresponding age-groups within the (18–37) age-range.

The decrease in mean heights beyond a certain age had been often ascribed to physiologic changes occurring after the individuals had reached skeletal maturity. When so exclusively explained, sight is lost of the secular trend of increasing height as a contributing factor, with the older age-groups being originally of lower stature.

In comparing heights by race, the white examinees appeared on the average between 0.1 and 0.4 of an inch taller than the Negro examinees. The greatest differences were in the younger age-groups.

Alongside with the mean heights, the standard deviations—measures of absolute variability, and the coefficients of variation—measures of relative variability—are shown in table 5. The functions of these values as an index of "clustering" about the mean are too known to require elaboration. For both white and Negro examinees, the standard deviations of height fluctuated around 2.6 or 2.7 inches, and the coefficients of variation around 3.8 and 4.0 percent. These values tend to increase with age, imparting increasing variability with advanced age. There were no material differences in the variability of heights, by race.

The modes of height, like its medians, proved to be practically the same as the corresponding mean heights. No separate modal values of height are therefore shown.

Weight. For white examinees, the mean weights by age were: 143.7; 149.3; 154.8; 156.7, and 157.3 pounds for the (18–19), (20–24), (25–29), (30–34), and (35–37) age-groups, respectively. For Negro examinees, the mean weights by age were: 141.8; 147.9; 152.0; 153.7, and 154.7 pounds for the same age-groups as given for white examinees. These data indicate an increase of 13.6 pounds in the mean weights of white examinees, and 12.9 pounds in those of Negro examinees, within the 20-year span. For both white and Negro examinees most of the increase was in the younger (18–29) age-groups.

White examinees under 25 years of age were on the average about 1.7 pounds heavier, and those 25 years of age and over were about 2.8 pounds heavier, than the Negro examinees of the same age-groups.

For white examinees, the standard deviations of weight were between 21 and 26 pounds, and their coefficients variation between 15 and 17%. For Negro examinees, the standard deviations were between 18 and 24 pounds, and their coefficients of variation between 12 and 15%. The white examinees thus show greater variability than the Negro examinees with respect to weight, whether measured on an absolute (standard deviation) or on a relative (coefficient of variation) basis.

TABLE 5

*Mean heights and weights of registrants examined for military service; standard deviations of these means and their respective coefficients of variation; also modes of weight, by race (January 1943 through January 1944)**

AGE AND RACE	HEIGHT			WEIGHT			
	Inches		C. V.	Pounds		C. V.	Modes
	Means	S. D.	(%)	Means	S. D.	(%)	(Pounds)
WHITE							
18–19	68.06	2.61	3.83	143.70	21.10	14.68	135.90
20–24	68.19	2.60	3.81	149.30	22.35	14.97	140.59
25–29	68.09	2.63	3.86	154.80	24.75	15.99	145.66
30–34	67.83	2.66	3.92	156.70	25.75	16.43	147.57
35–37	67.59	2.64	3.91	157.25	26.05	16.57	148.73
Total (18–37)	68.02	2.61	3.84	150.70	24.00	15.93	141.21
Total—Weighted by 1943-1944 U. S. White Male Population, Ages (18–37)	67.98	2.65	3.90	152.91	24.68	16.14	143.65
NEGRO							
18–19	67.64	2.57	3.80	141.85	17.45	12.30	137.84
20–24	67.91	2.65	3.90	147.85	18.40	12.45	143.26
25–29	67.90	2.64	3.89	151.95	20.80	13.69	145.47
30–34	67.70	2.66	3.93	153.70	22.70	14.77	145.36
35–37	67.51	2.70	4.00	154.70	23.85	15.42	145.74
Total (18–37)	67.76	2.66	3.93	149.40	20.80	13.92	142.56
Total—Weighted by 1943-1944 U. S. Nego Male Population, Ages (18–37)	67.78	2.66	3.92	150.37	21.26	14.14	143.40

* See footnotes to table 1-3; also "Appendix: Technical Notes," with respect to the computation of the "weighted values," and the "modes." S. D. = Standard Deviation; C. V. = Coefficient of Variation.

The most frequent (modal) weights are also presented in table 5 (last column), by age and race. These modal values differ greatly from the mean values caused by the skewed distributions of weight.

HEIGHT AND WEIGHT OF U. S. MALE POPULATION, AGE (18-37)

The mean heights of the examinees as a whole were 68.02 and 67.76 inches for the white and Negro groups, respectively. Their mean weights were 150.7 and 149.4 pounds (table 4).

In addition to these mean heights and weights and other height and weight values, corresponding values marked "weighted" are presented in the table for the whole age-range, by race. It was done because the examined registrants were preponderantly a young population when compared with U. S. male population within the same age-range, as may be observed from figure 1. The weighted values were computed by applying the age specific height and weight data of the examinees (table 4) to the distribution of the U. S. male population within the (18–37) age-range, by race. (See: "Appendix: Technical Notes," par. 4.)

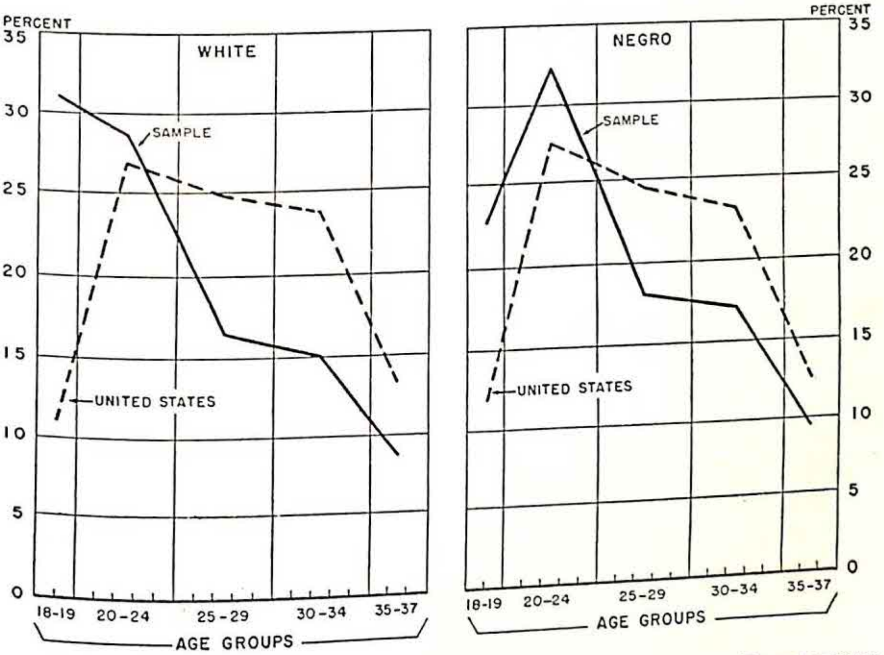

FIG. 1. AGE DISTRIBUTIONS OF SAMPLE AND UNITED STATES POPULATIONS. AGES 18 THROUGH 37, BY RACE, 1943.

These "weighted" values obviously reflect more closely the height and weight of U. S. male population within the (18–37) age-range, than the "unweighted" values given in the table for the examinees as a whole.

The differences between the "weighted" and "unweighted" values are primarily with respect to weight.

Based on these data, the following "weighted" mean heights and weights were computed:

a. White males (18–37) : Mean height—67.98 inches; mean weight—152.9 pounds.

b. Negro males (18–37): Mean height—67.78 inches; mean weight—150.4 pounds.[1]

DIFFERENTIAL VALUES

Height and weight values by region, size of community, and occupational group are presented in table 6. For some groups the mean heights and weights are above those of the total examined group; for others, they are below. But direct comparison of these differential mean values with total mean values may prove misleading since height and, especially, weight vary with age. Some of the differences in such comparisons might be due to age differences. To eliminate for comparative purposes the effect of age, "expected" mean values of height and weight were computed for each differential group, by race, as given in the table. These "expected" values indicate what would be the mean heights and weights of the particular differential group, if it had the age specific mean heights and weights of the total examined group. (For method of computation, see: "Appendix: Technical Notes," par. 5.)

The importance of comparing the mean values of the differential group with its "expected" mean values, instead of comparing them with the mean values of the total examined group, may be illustrated by the following, though extreme, examples. The mean weight of the white group of proprietors, managers, and officials, for instance, was 159.9 pounds, against a mean weight of 150.7 pounds for the total examined group. By contrasting these mean weights, the particular occupational group would be judged as being about 9 pounds heavier than the total examined group. Actually, the weight of this occupational group is expected to be greater since it is an older age-group—its mean age was 30.5 years as compared with 25.3 years for the total examined group (table 6). When the actual mean is compared with the "expected" mean, this occupational group shows a difference of about 5 pounds

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[1] In addition to this analysis by age and race, weight-height relationships were established by age and race, and standard weight-height tables were derived (Karpinos, '58).

(159.9 vs. 155.0 pounds)—one-half in magnitude of what it would be indicated by the former comparison. Another example, the white group of "no occupation" was on the average about four pounds lighter than the total examined group (146.8 vs. 150.7 pounds). This was all due to age differentials. Actually, its mean weight was slightly over its "expected" mean value (146.8 vs. 146.7 pounds).

Differential evaluation should, therefore, be made by contrasting the "actual" wtih the "expected" values, eliminating thus in such comparisons the influence of age-factors. It is also well to remember in connection with such appraisals that the factors responsible for such differences are interrelated. For instance, Northeast has greater concentration of larger cities than, say, South Central, which undoubtedly affects also its occupational distribution.

All height and weight values presented in table 6 have been adjusted for disproportionality, as explained above.

HEIGHT AND WEIGHT BY GEOGRAPHIC REGION

Height and weight values are presented in table 6 by the following geographic divisions: Northeast, Southeast, South Central, North Central, and West, by race. The states included in each of these regions are specified in footnote 2 to the table.

Height. In relation to mean height, the regions arrange themselves in the following ascending order:

a. White: Northeast (67.44 inches)—below its "expected" mean; North Central (68.15 inches), Southeast (68.21 inches), South Central (68.50 inches), and West (68.69 inches)—above their "expected" means.

b. Negro: Northeast (67.28 inches) and Southeast (67.71 inches)—below their "expected" means; North Central (67.74 inches)—the same as its "expected" mean; South Central (68.16 inches) and West (68.34 inches)—above their "expected" means.

These height data seem to indicate about the same regional pattern for both white and Negro examinees, with the Northeast region showing the lowest stature and the West the highest. For both white and Negro groups, the differences in height of these two extreme regions is somewhat over one inch. The data further suggest that where the white examinees were taller, the Negro examinees were also taller. The somewhat lower height noted above for the Negro examinees as a whole, when

TABLE 6

Heights and weights of registrants examined for military service, differentiated by: A. region, B. size of community, and C. occupation, separately for white and Negro

(January 1943 through January 1944)[1]

DIFFERENTIAL CLASS	MEAN AGE (Years)	HEIGHT (INCHES) Actual Means	Actual S. D.	C. V. (%)	Expected Means[3]	WEIGHT (POUNDS) Actual Means	Actual S. D.	C. V. (%)	Expected Means[3]
WHITE									
A. Region[2]									
Northeast	25.6	67.44	2.67	3.96	68.01	152.32	24.61	16.16	150.98
Southeast	25.0	68.21	2.51	3.67	68.02	147.30	23.15	15.73	150.44
South Central	24.8	68.50	2.70	3.95	68.03	147.06	23.83	16.21	150.20
North Central	25.3	68.15	2.54	3.73	68.02	152.04	24.10	15.86	150.72
West	25.1	68.69	2.70	3.93	68.02	151.06	22.93	15.19	150.54
B. Size of Community									
500,000 and over	26.2	67.63	2.69	3.97	68.01	153.30	24.94	16.28	151.53
100,000–499,999	25.8	68.05	2.69	3.95	68.00	151.28	24.60	16.27	150.29
10,000–99,999	25.4	68.07	2.63	3.87	68.01	151.28	24.37	16.12	150.80
2,500–9,999	24.9	68.19	2.58	3.79	68.02	150.07	23.75	15.83	150.34
Under 2,500	24.2	68.19	2.57	3.76	68.04	148.03	22.48	15.19	149.79
C. Occupation									
Professional	28.0	68.39	2.67	3.90	67.97	155.71	25.51	16.39	153.19
Proprietors, managers and officials	30.5	68.10	2.63	3.87	67.89	159.90	27.05	16.93	154.98
Clerks and kindred workers	26.2	68.11	2.62	3.85	68.00	151.61	24.82	16.38	151.56
Farmers and farm laborers	23.0	68.13	2.62	3.85	68.07	143.94	20.41	14.19	148.66
Skilled workers and foremen	26.7	68.10	2.62	3.84	67.99	153.98	24.71	16.06	152.06
Semiskilled workers	25.1	67.93	2.61	3.84	68.04	151.23	23.79	15.74	150.80
Unskilled workers	25.1	67.62	2.67	3.94	68.02	148.03	23.01	15.56	150.48
No occupation	21.4	68.28	2.69	3.93	68.06	146.82	22.49	15.33	146.71
D. Total Examined	25.3	68.02	2.61	3.84	68.02	150.70	24.00	15.93	150.70

NEGRO								
A. *Region* [2]								
Northeast	26.7	67.28	2.70	4.02	67.75	150.19	21.73	14.48	149.65
Southeast	25.6	67.71	2.65	3.91	67.77	148.94	20.09	13.49	148.98
South Central	26.6	68.16	2.62	3.84	67.75	148.81	20.70	13.91	149.58
North Central	27.0	67.74	2.61	3.85	67.74	150.79	22.56	14.97	149.87
West	27.0	68.34	2.59	3.79	67.75	151.97	24.22	15.94	149.94
B. Size of Community									
500,000 and over	27.2	67.46	2.65	3.93	67.75	150.13	22.13	14.75	150.08
100,000–499,999	27.0	67.72	2.66	3.93	67.76	149.48	21.78	14.58	149.89
10,000–99,999	26.4	67.78	2.65	3.90	67.77	149.64	21.04	14.07	149.54
2,500–9,999	25.6	67.95	2.63	3.87	67.77	149.24	20.57	13.79	148.90
Under 2,500	25.0	67.92	2.65	3.90	67.77	148.65	19.24	12.95	148.43
C. Occupation									
Professional	29.0	67.80	2.85	4.20	67.75	154.40	26.77	17.35	151.41
Proprietors, managers and officials	30.5	67.70	2.62	3.88	67.69	158.59	29.04	18.32	152.12
Clerks and kindred workers	25.9	67.43	2.67	3.96	67.76	146.69	22.03	15.03	149.02
Farmers and farm laborers	24.4	67.91	2.64	3.89	67.77	146.99	18.45	12.56	148.02
Skilled workers and foremen	27.4	67.87	2.64	3.88	67.77	151.85	21.70	14.29	150.30
Semiskilled workers	26.7	67.80	2.64	3.89	67.77	151.18	21.77	14.41	149.70
Unskilled workers	26.6	67.73	2.64	3.91	67.77	149.79	20.79	13.89	149.67
No occupation	25.0	67.58	2.70	3.99	67.75	145.79	20.20	13.87	148.16
D. Total Examined	26.1	67.76	2.66	3.93	67.76	149.40	20.80	13.92	149.40

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[1] See footnotes to table 5. S. D. = Standard Deviation; C. V. = Coefficient of Variation.

[2] States within each geographic region are as follows: *Northeast*—Connecticut, Delaware, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont; *Southeast*—Alabama, District of Columbia, Florida, Georgia, Kentucky, Maryland, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, West Virginia,; *South Central*—Arkansas, Louisiana, New Mexico, Oklahoma, Texas; *North Central*—Colorado, Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin, Wyoming; *West*—Arizona, California, Idaho, Montana, Nevada, Oregon, Utah, and Washington.

[3] See "Appendix: Technical Notes" for the computation of the "expected" means of height and weight.

compared with the height of white examinees, may be also noted when appraising their mean heights on a region by region basis.

Weight. In relation to mean weight, the regions array themselves in the following ascending order:

a. White: South Central (147.1 pounds) and Southeast (147.3 pounds)—below their "expected" means; West (151.1 pounds), North Central (152.0 pounds), and Northeast (152.3 pounds)—above their "expected" means.

b. Negro: South Central (148.8 pounds) and Southeast (148.9 pounds)— below their "expected" means; Northeast (150.2 pounds), North Central (150.8 pounds), and West (152.0 pounds)—above their "expected" means.

Both Southern white and Negro examinees seemed to be lighter in weight than those of the North and West. Within the Southern regions and the West, the Negroes seemed to be heavier than the white, while the opposite is true when a parallel comparison is made for the North, where the white appeared heavier than the Negroes.

The regional difference between the maximum and minimum mean weights were about 5 pounds for white examinees, and about three pounds for Negro examinees.

HEIGHT AND WEIGHT BY SIZE OF COMMUNITY

Differentiation by size of community (table 6) was made on the basis of the examinee's place of residence and its classification by size in the 1940 Census.

Height. The mean height seems to increase as one proceeds from the largest to the smallest communities. This is true of white and, to a lesser extent, of Negro examinees.

White examinees coming from cities over 500,000 inhabitants had a mean height about 0.4 inch below their "expected" mean height. Those from cities between 10,000 and 500,000 inhabitants approximated their "expected" mean heights, whereas white examinees from communities of less than 10,000 inhabitants had mean heights about 0.2 inch above their "expected" mean heights.

Negroes coming from cities over 100,000 inhabitants (the two largest city-groups) had mean heights below their "expected" mean heights. The difference is especially noticeable among Negroes from cities over

500,000 inhabitants; their mean height was about 0.3 inch below their "expected" mean height. As in the case of white, Negroes from the smaller communities (under 10,000 inhabitants) were above their "expected" mean height.

Weight. Weight seems to follow the opposite trend from that of height, namely it seems to decrease as one proceeds from the largest to the smallest communities. The decrease is especially noticeable in case of white examinees.

White examinees from the largest cities (500,000 and over) appeared to be by about 1.8 pounds heavier, when comparing their actual and "expected" mean weights (153.3 vs. 151.5 pounds). This holds true also for the next two city-groups: 100,000–499,999 and 10,000–99,999; their mean weights were above their "expected" mean weights, by one pound and one-half pound, respectively, (151.3 and 151.3 vs. 150.3 and 150.8 pounds). White examinees from the small communities (under 2500) were on the average about 1.8 pounds lighter than their "expected" weight (148.0 vs. 149.8 pounds). The last-mentioned groups of examinees consisted to a great extent of a farm population whose mean weight was far below their "expected" weight, as shown later in appraising weight by occupation. Examinees from the largest cities were on the average about four pounds heavier than those from the smallest communities.

No appreciable differences were found when similar comparisons by size of community were made for Negroes.

HEIGHT AND WEIGHT BY OCCUPATION

The examinee's occupation was coded in terms of the major occupational groups—a two-digit code adapted from the Classification Index of the Bureau of the Census, 1940. In the tabulations, however, the data were condensed into the well-known socio-economic groupings as given in table 6.

Height. Considerable variation in height was found among the various occupational groups, primarily among white examinees.

The following occupational groups of white examinees showed mean heights above their "expected" heights: professional persons; proprietors, managers and officials, and individuals of "no occupation." The last group consisted primarily of young people, as evidenced by their mean age (21.4 years). The mean heights of professional persons was 0.4

inches above their "expected" mean height, and the mean heights of the other mentioned occupational groups were 0.2 inches above their "expected" mean heights. The unskilled white workers, on the other hand, seemed to be relatively lower in stature; their actual mean height was about 0.4 inches below their "expected" mean height.

Among Negroes, no material variation in height was found by occupation, except for the clerks and kindred workers, and individuals of "no occupation." These two occupational groups had average heights of 0.3 and 0.2 inches below their "expected" mean heights.

Weight. Pronounced occupational differences were found in regard to weight. Among white examinees, proprietors, managers and officials, professional persons, and skilled laborers were on the average the heaviest. The mean weights of these occupational groups exceeded their respective "expected" mean weights by 4.9, 2.5, and 1.9 pounds, in order named. The average weights of white unskilled workers, and those of white farmers and farm laborers were below their "expected" weights: the mean weight of white unskilled workers was found as 2.5 pounds below their "expected" mean weights, and the mean weight of the white farmers and farm laborers as 4.7 pounds below their "expected" mean weight.

It should be mentioned with respect to the farmers and farm laborers that during this period the Selective Training and Service Act of 1940 was amended to provide for the deferment of registrants found to be "necessary and regularly engaged in an agricultural occupation or endeavor essential to the war effort." This well-known Tydings Amendment might have resulted in the exclusion of the higher economic groups within the farm population. Because of this selection, it is questionable whether the presented height and weight data for the farm and farm laborers could be taken as representative of that particular group.

Among the Negro examinees, proprietors, managers and officials, professional persons, skilled workers, and semi-skilled workers were on the average heavier than the total group. The mean weights of these groups exceeded their "expected" mean weights by 6.5, 3.0, 1.6, and 1.5 pounds, respectively. The other Negro groups (except the unskilled workers), namely farmers and farm laborers, clerks and kindred workers, and individuals with "no occupation" were on the average below their "expected" mean weights. Their mean weights were less than their "expected" mean weight by 1.0, 2.2, and 2.4 pounds, respectively. The mean weight of the unskilled Negro workers was practically the same as their "expected" mean weight.

COMPARISON WITH WORLD WAR I AND OTHER DATA

Overall Comparison: World Wars I and II. According to Davenport and Love ('21), the mean stature of the first million recruits of World War I was 67.49 inches, and their mean weight was 141.54 pounds (table 7). Like those of World War II, these measurements were taken with the recruits stripped.

TABLE 7

Comparison of height and weight of army inductees, World Wars II and I, by region

GEOGRAPHIC REGION [1]	HEIGHT (INCHES)			WEIGHT (POUNDS)		
	WW II	WW I [2]	Gain	WW II	WW I [2]	Gain
Total	68.07	67.49	0.58	150.45	141.54	8.91
Northeast	67.55	66.74	0.81	152.04	139.37	12.67
Southeast	68.14	67.92	0.22	147.57	140.81	6.76
South Central	68.58	68.19	0.39	147.54	141.60	5.94
North Central	68.20	67.63	0.57	151.63	142.77	8.86
West	68.74	67.85	0.89	151.19	144.67	6.52

[1] See footnote 2, table 6.

[2] Derived from tables 13 and 30, Davenport and Love ('21).

The term "recruit," as was employed in World War I, referred to individuals examined at military camps after they had been screened out by the local boards. During World War I, local board screening was of primary nature and was not restricted to eliminating men with manifestly disqualifying defects as was the case during the period to which our data of World War II relate. It is, therefore, appropriate to compare the height and weight of World War I recruits with those of World War II inductees, as done in table 7.

A direct comparison of these height and weight data indicate that the World War II inductees were on the average 0.6 inch taller (68.07–67.49) and 8.9 pounds heavier (150.45–141.54) than the World War I recruits. Actually, however, the differences in these measurements are larger than those indicated by this comparison, since the inductees of World War II were on the average younger than the recruits of World War I. When due allowance is made for the age differences of the inductees of the two considered periods, the age-adjusted height and weight values would indicate that within the same age-range the inductees of World War II were about two-thirds of an inch taller and about 10.7

pounds heavier than those of World War I. (The adjustment for age was made by applying the present age specific height and weight values to the age distribution of the World War I recruits as given in Davenport and Love, '21, table 2. See: "Appendix: Technical Notes," par. 5.)

World War II data indicate less variability with respect to height—the standard deviation for height were 2.62 and 2.71 inches for World War I and II, respectively, but greater variability with respect to weight—the respective standard deviations for weight were 22.1 and 17.4 pounds. The height and weight distribution of World Wars I and II are graphically depicted in figure 2. The distribution of World War II examinees by weight shows a wider scatter toward higher weights than that of World War I.

Regional Comparison: World Wars I and II. Davenport and Love ('21) noted geographic (state) differences in height and weight data of World War I. Since the present data were not available by state, the state data of World War I were combined for comparison into the geographic regions used for World War II data. The World War I data by regions are presented in table 7, alongside with the corresponding World War II data for inductees. The gains in height and weight between World Wars I and II are also shown in table 7, by region.

In discussing the geographic differences in height in World War I, Davenport and Love ('21) attributed these in part to regional differences in the influx, shortly before World War I, of immigrants from southeastern Europe, namely, of men of relatively low stature. For instance, they offered this immigation factor, in particular, and the high proportion of certain foreign-born men in the population, in general, as an explanation for the relatively lower stature of recruits from the Northeast region. These factors appeared to be still operative to a certain extent in World War II.

As may be seen from table 8, the proportions of foreign born white males of military age were markedly decreased during the periods close to World Wars I and II. Between 1920 and 1940, the precent of foreign born white males declined from 18.5 to 4.4, within the (20–34) military age-range. However, these proportions still remained relatively high in World War II in Northeast, West, and North Central regions, though in absolute terms the largest decrease in the proportions of foreign born occurred within these regions. It is within these regions that the largest gains in height occurred between World Wars I and II.

The gains in height by regions were as follows in ascending order:

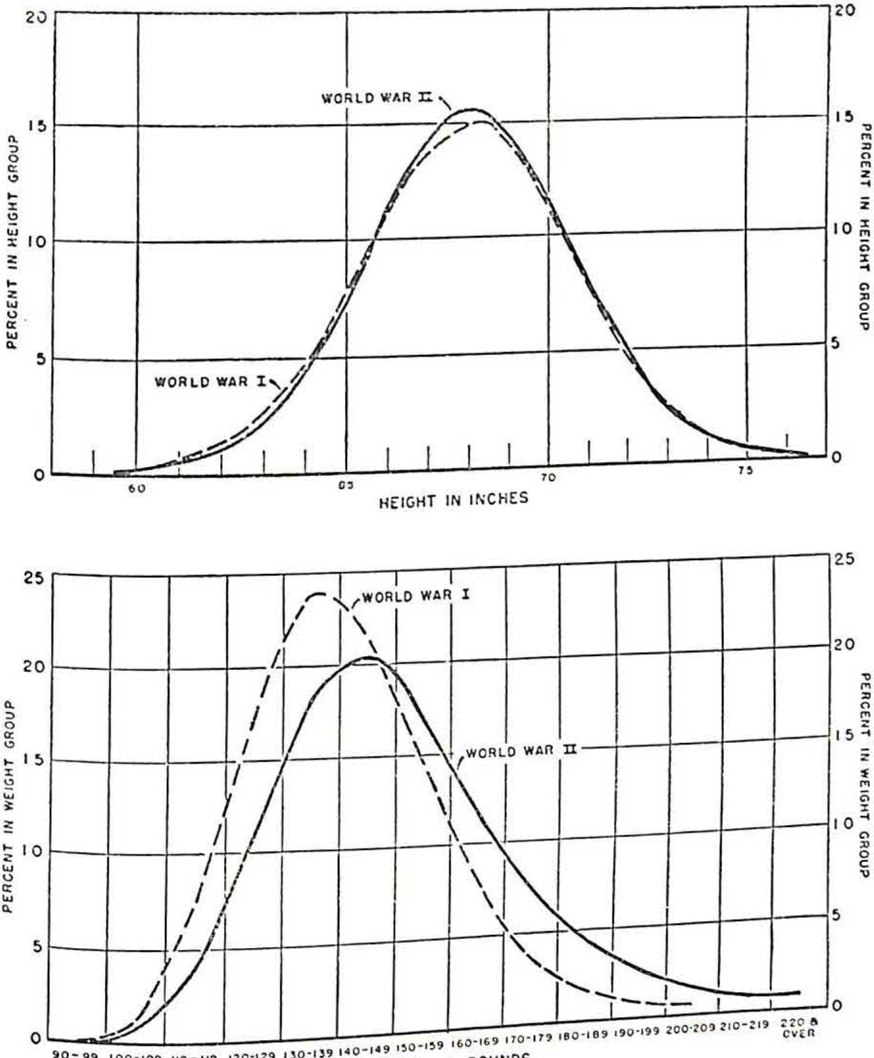

FIG. 2. PERCENT DISTRIBUTION OF HEIGHTS AND WEIGHTS. ARMY INDUCTEES, WORLD WAR II, COMPARED WITH RECRUITS, WORLD WAR I.

Southeast—0.22; South Central—0.39; North Central—0.57; Northeast—0.81, and West—0.89 inches (table 7). This suggests that the gains in height may at least in part be ascribed to an increase in the homogeneity of the population-composition between World Wars I and II.

The gains in weight by region were as follows, in ascending order: South Central—5.9; West—6.5; Southeast—6.8; North Central—8.9,

TABLE 8

*Percent of foreign born white males, ages 20 through 34, as of total white males of the same ages, by geographic region (1910-1950)**

GEOGRAPHIC REGION	PERCENT FOREIGN BORN 1910	1920	1930	1940	1950
Total	24.2	18.5	11.4	4.4	2.5
Northeast	27.7	24.5	22.0	17.8	14.0
Southeast	3.1	2.7	2.1	1.7	1.7
South Central	5.9	6.3	4.8	2.9	2.9
North Central	17.8	15.2	12.6	9.2	6.7
West	24.5	20.8	17.8	12.3	8.9

* Computed from various Census reports. See footnote to table 6 for the states within each geographic region.

and Northeast—12.7 pounds. Part of these gains in weight between World Wars I and II may be attributed to increasing height. Mostly, however, it is apparently due to improved nutrition—both quantitatively and qualitatively. Further investigations are required to explain the wide differences found in the gains of weight by region.

Other Comparisons. Further comparable—though fragmentary—

TABLE 9

*Comparative height and weight data **

INDUCTEES OR RECRUITS	HEIGHT (INCHES) Mean	S. D.	WEIGHT (POUNDS) Mean	S. D.
U. S. Army Inductees, World War II:				
Age (18–19)	68.12	2.56	144.56	19.77
Age (20–24)	68.20	2.58	149.51	21.00
English Recruits (1939), Age (20–21)	67.50	2.62	135.70	16.54
Royal Air Force Personnel (1948):				
Age (18–19)	67.52	2.52	132.15	15.26
Age (20–25)	68.17	2.54	138.58	15.01
English Army Intake (1951):				
Age (18–19)	67.77	2.62	137.37	16.68
Age (20–24)	68.16	2.59	143.09	17.20

* Sources: English Recruits (1939): Martin ('49); Royal Air Force Personnel: Morant ('49), Tables I and II (All Stations), at the beginning of training; English Army Intake (1951): Rosenbaum ('54), Tables 1 and 5.

height and weight data are presented in table 9. The data are for English recruits for the following periods: 1939, 1948, and 1951.

The U. S. inductees of World War II appear on the average somewhat taller and much heavier than the English recruits either of 1939 or 1948, and even of 1951. The English instructions for taking the height and weight measurements require that men wear trousers and socks. Corrections of one-quarter inch for height and from two to three pounds for weight are usually made in comparing these measurements with those taken in the nude (Rosenbaum, '54). Hence, U. S. inductees seemed to be on the average about one-half inch taller and 10–14 pounds heavier than the English recruits.

HEIGHT AND WEIGHT OF REGISTRANTS WITH TUBERCULOSIS

Except for registrants who were disqualified for tuberculosis, no attempt was made to relate the height and weight data to any disease or defect. The mean heights of the tuberculous registrants were as follows: White—68.29 inches; Negro—67.90 inches, exceeding the mean heights of all examinees by 0.27, and 0.14 inches, respectively. Analogous differentials were noted by Davenport and Love ('21) for World War I, who reported the average stature of recruits with pulmonary tuberculosis as 0.58 inch above the mean height of all recruits. The mean height of the tuberculous patient of World War II was about the same as that of World War I (68.1 inches—World War I). (See Long and Jablon, '55, p. 82, on the relationship of tuberculosis and constitutional factors.)

With respect to weight, the following averages were found for tuberculous registrants in World War II: white—146.9 pounds; Negro—145.0 pounds. These figures indicate for white and Negro tuberculous registrants mean weights of 3.8 and 4.4 pounds below the mean weights of the examined groups. According to Davenport and Love ('21), the average weight of recruits with tuberculosis in World War I was 11 pounds below the average of all recruits.

In spite of the fact that the tuberculous registrant of today is about four pounds lighter than the average registrant, his mean weight is about five pounds above the mean weight of the recruit of World War I, and about 16 pounds above that of the recruit of World War I with tuberculosis. (The average weight of the World War I recruit with pulmonary tuberculosis was 130.4 pounds.) These data exclude institutionalized cases of tuberculosis.

SUMMARY

1. The findings presented here are based on an analysis of height-weight data abstracted from about half-million physical examination forms (DSS Form 221). These forms were selected at random from about five million forms received at the Surgeon General's Office, Army, on Selective Service registrants processed for military service in World War II during the thirteen-month period—from January 1943 through January 1944. These registrants were within the age limits of 18 through 37, but primarily (about 40%) concentrated in the under 21 age-group.

2. Among both white and Negro examinees, the (20–24) age group—age of maturity—had the highest stature: 68.19 and 67.91 inches, respectively. The mean heights of the (18–37) groups, as a whole, were computed as 68.02 and 67.76 inches, for white and Negro examinees, respectively (table 5).

3. With respect to weight, their means ranged from 143.7 to 157.3 pounds for white examinees, and from 141.8 to 154.7 pounds for Negro examinees, within the 20-year period: from (18–19) through (35–37) age-groups. The mean weights for these examinees, as a whole, were computed as 150.7 and 149.4 pounds, for white and Negro examinees, respectively (table 5).

4. The Negro examinees appeared somewhat shorter and much lighter than the white examinees (table 5).

5. Differences in height and weight were found among examinees from the various geographic regions. Examinees from the West and South Central regions proved to be taller than those of other regions. On the other hand, examinees from Northeast and North Central were the heaviest (table 6).

6. Examinees from the largest cities appeared to be shorter and heavier and those from the smallest communities taller and lighter, than all examinees (table 6).

7. Examinees higher on the socio-economic scale appeared relatively taller and heavier (table 6).

8. Compared with the recruits of Word War I, the inductees of World War II were found about 0.6 inch taller and about 9 pounds heavier (table 7). When adjustments were made for the differences in their age distributions, the inductees of World War II seemed to be on the average two-thirds of an inch taller and by about 10.7 pounds heavier

than the recruits of World War I. The distribution of the examinees of World War II by weight shows a greater scatter towards heavier weights than that of World War I (figure 2).

9. The data indicate marked differences among regions in the gains of height and weight between World Wars I and II. The greatest increase in height occurred in the West and Northeast; the greatest increase in weight took place in Northeast and North Central (table 7).

10. Comparison between U. S. World War II inductees and English recruits indicate that the U. S. inductees of World War II were about one-half inch taller and 10–14 pounds heavier than the English recruits of that period (table 8).

11. Tuberculous registrants were found taller and lighter when compared with all examinees.

APPENDIX: TECHNICAL NOTES

1. *General.* As pointed out in the text, about one-fourth (23.6%) of the qualified registrants were inducted during this period into the Navy. No provision was made to furnish the Surgeon General's Office, Army, with copies of the physical examination forms of the Navy inductees. The qualified group included in this study was hence limited to Army inductees, resulting in a partial coverage of this group.

Since intergroup (inducted vs. qualified) as well as intragroup (age) differences were found with respect to the height and weight values, it became evident that the height and weight values for the total examined group cannot be obtained by directly combining the height and weight data of the disqualified with those of the inducted examinees. Such a procedure would have given undue proportionality to the disqualified group. To correct for this disproportionality, determination had to be made first as to whether the Army inductees could be taken as representative of all inductees with respect to height and weight.

There were some differences in the manner of assigning qualified registrants to the Army or the Navy. For one thing, qualified registrants classified as "limited service" could not be assigned to the Navy. However, a study of the "limited service" inductees showed that the distributions of these inductees by height and weight were essentially the same as those of the other inductees. For another, qualified registrants, excluding "limited service," were further classified as "general service," acceptable for both the Army and the Navy, and "special assignment" who were acceptable for the Army alone until June, 1943. The latter group of registrants included acceptable illiterates and registrants who met the Army's visual and hearing standards for general service, but not those of the Navy (Surgeon General's Office, '44). Judging from the composition of the "special assignment" group, there was no reason to suppose that the height and weight data of this group could have been materially different from those of the "general service" group. Furthermore, the number of registrants in the "special assignment" group was relatively small, in addition to the fact that the restriction with respect to their assignment to the Navy was effective only for the first five months of the thirteen-month period under consideration.

However, there was a third difference directly related to height and, hence, to weight. While the acceptable height-range for Army inductees was between 60 and 78 inches, that of the Navy was restricted to 62 through 76 inches. Therefore, the adjustments were made in the cross-tabulations of the Army inductees by height and weight within the height-range of 62 to 76 inches.

2. *Adjustment Factors.* In addition to the copies of the physical examination reports (DSS Form 221) forwarded to the Surgeon General's Office, Army, each induction station was required to submit a monthly report ("Monthly Report of Examinations at Joint Army and Navy Induction Station,") relating to the general results of these examinations. This report showed for each Army Area and State, by race: the number examined; number disqualified, distributed by the principle disqualifying cause, and number of inductees, by type of qualification (general service, special assignment, and limited service), and department of assignment: Army or Navy (Surgeon General's, '44).

On the basis of the data obtained from these monthly reports, the ratios of qualified to disqualified registrants were established, by race. The adjustment factors for the qualified groups were thus determined as 1.32, and 1.11 for white and Negro inductees, respectively.

Those adjustment factors were applied to each age-group within the particular qualified group, since age was not a selective factor in the assignment of the Army or the Navy. Each cross-tabulation of the Army inductees by height and weight was adjusted within the height-range of 62 to 76 inches. Each adjusted cross-tabulation of the qualified registrants was then combined with the corresponding cross-tabulation of the disqualified registrants to obtain the proper cross-tabulations for the total examined.

3. *Determination of Modes.* The modes were computed by utilizing four moments (Croxton and Cowden, '55, p. 190, footnote 5).

4. *Height and Weight Values for U. S. Male Population (18–37).*

a. General. Since height and, particularly, weight differ with age, and since the distribution by age of the U. S. Male population within the age range of (18–37) is much different from that of the total examined group, as may be observed from figure 1, the various values (means and standard deviations) for total U. S. male populations, aged 18–37, had to be obtained by weighting the specific age values of the examined group by the age distribution of U. S. male population, by race. The procedures applied for computing these required "weighted" values are explained in *b*, below.

b. Weighted Means and Weighted Standard Deviations. Let $\bar{X}_i$ and $\bar{Y}_i$ represent the respective means of height and weight of any age-interval within any examined group, while $\bar{X}$ and $\bar{Y}$ the corresponding mean values of the combined group.

Obviously, $$\bar{X} = \sum w_i \bar{X}_i \qquad (1),$$

where w_i is the proportion of any age group within the total group ($\sum w_i = 1.000$). For U. S. male population (18–37), w_i represents its proportional age distribution within this age range, which was determined as follows on the basis of supplied Census data:

White: Under 20—.1070; 20–24—.2699; 25–29—.2490;
30–34—.2389; 35–37—.1352 ($\sum w_i = 1.0000$),

Negro: Under 20—.1169; 20–24—.2806; 25–29—.2443;
30–34—.2337; 35–37—.1245 ($\sum w_i = 1.0000$).

The corresponding standard deviations were derived on the basis of the following relationship of the variance:

$$V(X) = \sum w_i [V(X_i) + \bar{X}_i^2] - \bar{X}^2 \qquad (i = 1, \cdots, k) \qquad (2),$$

where $V(X)$ represents the variance of the total U. S. male group; $V(X_i)$—the variance of the particular age group; $\bar{X}_i$ and $\bar{X}$, as in (1), above. The same holds, of course, for Y. (See Kenney, '47, corollary 1, p. 101.)

5. *"Expected" Values.* The "expected" values given in table 6 were computed by what is known as the "indirect" method of standardization. Briefly stated, this method is used in comparing values of two or more groups whenever these groups differ with respect to some variables, say age, but the specific values by this variable are unknown. In our case, the average height and weight are known for each differential group (table 5), as a whole, but not by age. A direct comparison of the mean height and weight of any differential group with those of all examinees would hardly be justified since these groups differ considerably in age, while height and, especially, weight are a function of age. The indirect method of standardization is used in order to eliminate the effects produced by age differences. It was accomplished here by multiplying the age specific height and weight values of the total examined group by the age distribution of the particular differential group. (Age distribution of each differential group was available.) The obtained values are the "expected" values. The differences between the actual values of differential group and its "expected" values reflect actual differences, not affected by age. (See: Stouffer, '51, for a detailed discussion in regard to this method of standardization.)

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SOME INTERRELATIONS BETWEEN SOMATIC, ROENTGENOGRAPHIC AND DENSITOMETRIC CRITERIA OF FATNESS[1]

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AN individual's leanness-fatness may be estimated on the basis of several procedures, varying in the complexity of manipulations and underlying assumptions (Keys and Brožek, '53). Here we have considered three procedures: caliper measurements of skinfold thickness, measurements on soft-tissue roentgenograms of the extremities, and body density determinations.

Interrelations between these approaches were examined from three points of view:

1) Using body density as a criterion of the fat content of the body as a whole, correlations with the thickness of subcutaneous adipose tissue were computed. They represent a measure of the *validity* of linear somatic and roentgenographic measurements as indicators of total body fat. In addition, the usefulness of employing absolute or relative measures of the layer of subcutaneous adipose tissue, taking into account body size, was examined.

2) Correlations between skinfolds and roentgenographic widths indicate the *reliability* of the two procedures which aim to measure the same thing, i. e. the thickness of the *panniculus adiposus*.

3) In addition to the assessment of the degree of association among

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the variables under study, prediction equations were calculated in order to make possible comparisons of data obtained by the three procedures and, in particular, to provide means for converting the roentgenographic values into densitometric estimates of total body fat. Such equations had been provided for skinfolds (Brožek, '54; Pascale *et al.*, '56), even though additional work in this direction is necessary in order to take into account more adequately the variables of sex and age as well as the advances in the techniques of measurement and their standardization (Committee, '56).

This presentation is focused on the relations between density, considered as indicator of total fat content of the body, and the roentgenogrammetrically determined thicknesses of subcutaneous fat. Data were provided for the empirical appraisal of the value of absolute thicknesses vs. thicknesses related to various criteria of body size.

METHODS AND PROCEDURES

In determining body density, defined as the ratio of mass and volume, body volume was obtained by underwater weighing. The water temperature was kept at 36 ± 0.5°C. The air remaining in the lungs and respiratory passages at the end of maximal exhalation (i.e. at a moment when the weight under water was read) was determined for each individual by the nitrogen-dilution technique and the value was subtracted from the gross volume of the body (Brožek, *et al.*, '49).

Skinfold thickness was measured at the back of the right upper arm, over the triceps. The level at which the measurement was made was defined as halfway between olecranon and acromion, and was marked with a dermatographic pencil. The level of the upper arm, one-third of the distance up from the olecranon, was also marked for positioning the upper arm for soft-tissue roentgenograms (Site No. 3, see below). In making the skinfold measurements the caliper was applied with a pressure of 10 gm/mm² of the circular contact surface (25 mm²).

Soft-tissue roentogenograms were taken at a tube-to-film distance of 72 inches. The following sites were used, for exploratory purposes, on the right extremities:

1) Upper arm, in anteroposterior (A-P) projection, at the level of deltoid insertion.

2) Upper arm, in medio-lateral projection (M-L), half-way between olecranon and acromion, at a level at which the skinfold thickness was

measured. The dorsal roentgenographic fat measurement corresponds to one-half of the uncompressed skinfold.

3) Upper arm, in A-P projection, one-third of the distance between olecranon and acromion.

4) Forearm, in a position of supination (palm of the hand facing upwards), A-P projection, at the level of maximal width.

5) Thigh, one-third of the distance between the lower border of the patella and the anterior superior iliac spine, A-P projection. Because of the size of the casette used, only the medial thickness of subcutaneous layer was considered.

6) Calf, in A-P projection, at the level of maximal width.

The measurements of the thickness of the adipose tissue layer, muscle and bone were made as follows:

At the point of deltoid insertion (Site No. 1) the thickness of the layer of skin and subcutaneus tissue was measured along the line vertical to the skin (*cf*. Garn, '54, who used on P-A projection).

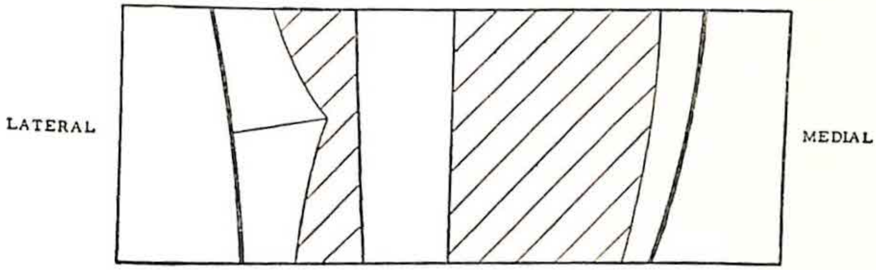
At sites No. 4 and 5 (forearm and calf) the measurements of adipose layer, bone and muscle or bone plus muscle (in Site No. 4) were taken perpendicularly to the long axis of the limbs (line connecting the middle points of the upper and lower edges of the section of a limb), at the level of the widest diameter.

At Sites No. 2, 3, and 5 (upper arm and thigh) the measurements of soft tissues were made along the line perpendicular to the long axis of the limb, same as in Sites No. 4 and 6. Bone width was measured perpendicularly to the long axis of the bone.

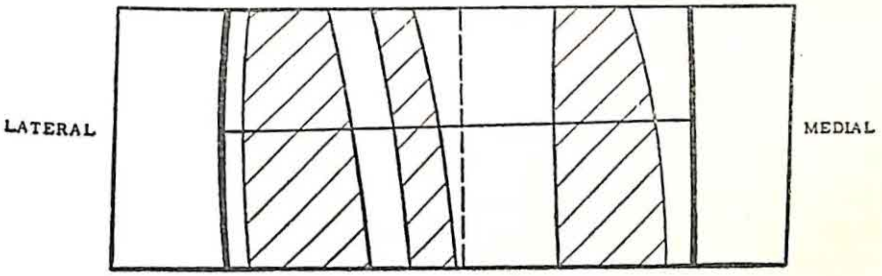
Fig. 1 shows three examples of the method of measurement. For purposes of comparison with data reported in the literature, the dorsal fat layer in Site No. 2 (mid-upper arm) was measured also vertically to the skin.

SUBJECTS

The subjects were drawn from a group of middle-aged business and professional men participating in a longitudinal study of aging of the cardio-vascular system. The need for selection resulted from the fact that the densities were determined in 1951 while the soft-tissue X-rays were taken in 1955. Skinfold measurements were made on both occasions. The present sample consists of men whose 1955 weight did not differ by more than ± 2% from the 1951 weight. In one-half of the men the weight changes were within ± 1% while in the other half the body weight varied by more than ± 1% but did not exceed ± 2%.



DELTOID INSERTION



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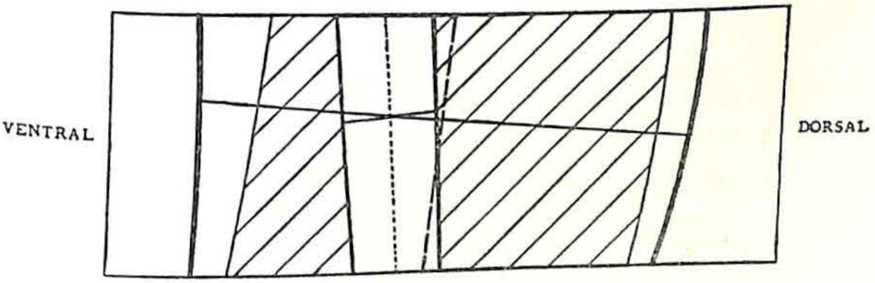

MID-UPPER ARM

FIG. 1. TECHNIQUE OF MEASURING SOFT-TISSUE ROENTGENOGRAMS. LIMB AXES ARE INDICATED BY LONG DASHES, BONE AXIS BY SHORT DASHES. THE LINES ALONG WHICH THE MEASUREMENTS WERE MADE ARE SOLID.

The general characteristic of the sample, referring to the year 1955, are given in table 1. The age range was quite narrow while the variability in relative body weight, obtained by referring the actual weight to the tabular age-height-weight standards, was very close to that found in the population from which the subsample was drawn (Brožek and

TABLE 1

Means and standard deviations of the general characteristics of the sample ($N = 52$)

	MEAN	SD
Age, years	57.1	2.7
Height, cm.	175.9	6.7
Weight, kg.	80.2	10.4
Relative body weight, %	104.3	12.2

Keys, '51, p. 200). The mean actual weight exceeded slightly the reference weight but it should be pointed out that the height-weight tables used (Association, '12) do not go beyond the age of 55 and, consequently, values of relative body weight of adults in the upper age range are only approximate. The fact that the subjects were weighed in light trunks, while the insurance applicants on whom the reference tables are based were weighed in indoor clothing and with shoes on, further stresses the approximate character of the reference values a criterion of "normality" of weight.

TABLE 2

Medians, means and standard deviations of the 3 types of fatness indicators (Thicknesses are given in mm)

CHARACTERISTIC	MEDIAN	MEAN	*SD*	*N*
Body density	1.0392	1.0401	0.0124	52
Skinfold thickness				
Upper arm, ½ up, dorsal	13.2	13.0	4.1	52
X-ray measurements				
Deltoid insertion	18.0	17.9	3.6	42
Upper arm, ½ up, dorsal	7.8	7.9	2.4	52
Upper arm, ½ up, dorsal & ventral	15.8	16.4	4.8	52
Upper arm, ⅓ up, medial & lateral	17.9	17.8	4.2	41
Forearm, max. width, medial & lateral	10.9	10.9	3.0	51
Thigh, ⅓ up, medial	17.4	17.4	4.4	42
Calf, max. width, medial & lateral	11.0	11.4	3.3	47

The medians, means and standard deviation of body density, skinfold thickness and the thicknesses of skin-plus-subcutaneous layer measured on roentgenograms are given in table 2. The distributions were only moderately asymetrical. Body density, converted into total body fat by using the 1953 equation of Keys and Brožek ('53), indicates that on the average 22.6% of body weight may be accounted by body fat. In comparing the average skinfold with data reported in the literature the pressure used (10 gm/mm^2 in this study) should be taken into account.

INTERRELATIONS BETWEEN MEASUREMENTS

On the average, half of the skinfold is 6.5 mm, corresponding to 82.3% of the roentgenogrammetric value of skin plus subcutaneous layer (table 2), measured at approximately the same place. The difference is due largely to the compression of the skinfold. The caliper pressure is, clearly, a crucial factor. There may be regional and other (sex, age) factors that may modify the degree of effective compression. These problems require further research.

A small amount of the difference between skinfolds and X-ray measurements is contributed by the magnification of the roentgenographic values as a result of the fact that the rays, even at a focal-film distance of 6 feet, are slightly divergent, not parallel, and the median plane of the limbs is separated from the film plane. The average magnified arm measurements were about 102.5% of the "true" value.

The equations relating the three types of measurements of leanness-

TABLE 3

Equations ($Y = a + bX$) *interrelating body density, skinfold thickness (upper arm, ½ up, dorsal) and the dorsal width of the subcutaneous layer of upper arm measured on roentgenograms at approximately the same site*

Y	X	a	b
Body Density	Skinfold (mid-upper arm, dorsal)	1.0672	—0.0021
Body Density	X-ray width (mid-upper arm, dorsal)	1.0655	—0.0032
Skinfold (mid-upper arm, dorsal)	X-ray width (mid-upper arm, dorsal)	1.55	+1.44
X-ray width (mid-upper arm, dorsal)	Skinfold (mid-upper arm, dorsal)	1.83	+0.47

fatness are given in table 3. In calculating individual values of body density only three decimal figures are significant and the calculated values should be so rounded. Similarly, the predicted figures of subcutaneous fat should be rounded out to the tenth of a millimeter or to the nearest half-millimeter (for skinfolds measured with this precision).

The closeness of the association is indicated by the coefficients of correlation (table 4). It is of interest to note that the thickness of the dorsal skinfold does not show a lower correlation with total body fat than the corresponding roentgenographic measurement, as might perhaps be expected. The coefficient of correlation between the two approaches (somatometric and roentgenogrammetric) to the measurement of subcutaneous fat is similar to the values reported in the literature.

TABLE 4

Coefficients of correlation between body density, dorsal skinfold thickness, and dorsal width of subcutaneous layer of upper arm measured on roentgenograms

X	Y	r	N
Density	Skinfold	—0.68	52
Density	X-ray width	—0.63	52
Skinfold	X-ray width	+0.82	126

Coefficients of correlation between body density and combined—dorsal and ventral, lateral and medial—roentgenographic measurements tend to be usefully higher (table 5, last column) than when single measurements are utilized. The only exception is the calf in which the lateral width is a substantially poorer indicator of total fat than the medial width. Equations for predicting body density from roentgenographic data are given in table 6.

TABLE 5

Cofficients of correlation between body density and separate versus combined (Dorsal and Ventral, Lateral and Medial) roentgenographic measurements of the thickness of the subcutaneous fat layer (plus skin)

Upper arm, middle	—.63 (D)	—.67 (V)	—.73 (D & V)
Upper arm, $\frac{1}{3}$ up	—.64 (L)	—.62 (M)	—.75 (L & M)
Forearm	—.67 (L)	—.74 (M)	—.76 (L & M)
Calf	—.34 (L)	—.66 (M)	—.58 (L & M)

TABLE 6

Equations for predicting body denstiy ($\hat{Y}$) *from roentgenographic measurements,* X, *in mm, in middle aged men* ($\hat{Y} = a + bX$)

X	a	b
Deltoid insertion, (Upper arm)	1.0722	—0.0019
Upper arm (midway, dorsal + ventral)	1.0712	—0.0019
Upper arm (lower third, medial + lateral)	1.0781	—0.0022
Forearm (maximum width, medial + lateral)	1.0729	—0.0031
Thigh (lower third, medial)	1.0624	—0.0014
Calf (maximum width, medial + lateral)	1.0645	—0.0023

The data on actual versus size-corrected measurements of roentgenographic widths of subcutaneous adipose tissue (plus skin) are presented in Table 7. The first column refers to the actual, uncorrected measure-

TABLE 7

Coefficients of correlation between body density and absolute (X) *or relative measurements of subcutaneous fat (as ratios to stature,* L*; to the corresponding bone width of the limb,* B*; and to bone plus muscle width,* $B + M$)

	X	X/L	X/B	$X/(B + M)$	N
Deltoid insertion, upper arm	—.60	—.66			
Upper arm, middle, dorsal	—.63	—.64	—.64	—.63	52
Upper arm, middle, dorsal and ventral	—.73	—.75	—.76	—.73	52
Upper arm, lower third, medial + lateral	—.75	—.75	—.74	—.68	41
Forearm, max. width, medial + lateral	—.76	—.77		—.79	51
Thigh, lower third, medial	—.51	—.52	—.51		42
Calf, max. width, medial + lateral	—.58	—.59	—.57	—.56	47

ments. Relating the roentgenographic fat measurements to various indicators of "size"—stature, width of the bone measured at a given level of the limbs, or combined width of bone and muscle—does not substantially increase the size of the coefficients of correlation with body density. Only the consideration of body length resulted in consistently higher coefficients. However, even here the magnitude of the differences was negligible.

It should be pointed out that the coefficients of correlation between roentgenographic measurements and density are somewhat reduced because of changes in weight (and fatness) that took place between 1951, when the body densities were determined, and 1955, when the soft tissue roentgenograms were taken. Fortunately, skinfold measurements were made in both years and the magnitude of the difference due to this source of variability may be estimated. The coefficients of correlation with body density were —0.683 using the 1955 skinfolds ($N = 52$), and —0.723 using the 1951 skinfolds ($N = 52$). For the group as a whole ($N = 160$), the r obtained for 1951 values of both density and skinfolds was —0.726.

DISCUSSION

Single sites versus combined measurements

Roentgenographic measurements of subcutaneous fat may serve one of two, diametrically opposed, goals: 1) One may wish to describe the patterns of distribution of subcutaneous fat (*cf.* Garn, '55, p. 537) and to bring out the *regional differences* in fatness by contrasting, *e.g.*, the fat depots on the extremities and those on the trunk, or 2) one may be interested in arriving at the best estimate of *total* fat, i. e. of the "fatness" of the organism rather than the fatness of particular regions (*cf.* Garn, '57).

The latter purpose is served best by combining the separate measurements in the form of multiple regression equation, as has been done for skinfolds (Brožek and Keys, '51). This technique provides means for optimal weighing of the measurements made at different sites and for the elimination of those variables that do not contribute significantly to the precision of the prediction. Neither of these important features is present when the single measurements are simply added (or averaged). In the present series the number of individuals for whom all of the required data were available was considered too small for the task of combining the single sites in the form of a multivariable prediction equation. This is, clearly, an urgent task for future research.

Effect of size

The problem of absolute (actual) and relative values of subcutaneous adipose tissue as indicators of total body fat is intriguing. It has been a fairly general practice, in adults at least, to use the actual (absolute) measurements as a criterion of leanness-fatness. On theoretical grounds, considerations of body size would appear to be called for. The available evidence, however, is essentially negative.

We have seen (table 5) that relating—in the form of an index—the subcutaneous layer to stature, width of the bone of the extremity, or to the width of bone plus muscle, has only increased negligibly or has even depressed, in the case of bone and bone-plus-muscle used as standards of reference, the correlation with body density.

This is in line with our previous experience with absolute and relative skinfolds. We were unable to demonstrate (Keys and Brožek, '53, p. 262) any gain in the precision of prediction of body density from skinfolds when surface area (based, in its turn, on height and weight) was taken into account as a measure of body size. The correlations between height and the thickness of subcutaneous layer of adults tend to be close to zero whether one uses roentgenographic data (*cf.* Reynolds, '50, p. 69, values for the age level of 15.5 years) or skinfold (Brožek, '56, p. 137).

In this study the sample of adults was of the same sex and highly homogeneous in age. In comparing the relative fatness of the sexes, Reynolds ('49) found useful the fat/bone index. It reflects the fact that females not only tend to have larger absolute breadths of subcutaneous fat (19.7 vs. 9.6 mm at the calf) but their bone breadths are smaller (32.8 vs. 39.5 mm; *cf.* Reynolds and Groote, '49, p. 47). Under these conditions, a relative fat breadth shows the difference in fatness more distinctly than the absolute fat breadths.

Prediction of total body fat

The prediction equations (tables 3 and 6) have primarily a practical value, enabling one to "translate" one set of measurements into another. This applies especially to the relations between skinfolds and roentgenographic values of subcutaneous fat. Predictions of body density are of greater interest inasmuch as they make it possible to estimate the total amount of body fat and thus yield information needed in studies in which a fat-free weight is sought as a reference point in metabolic and physiological investigations.

The derivation of the equation for calculating body fat (F), as percentage of body weight, from body density (D)

$$F = 100[(4.201/D) - 3.813]$$

has been given elsewhere (Keys and Brožek, '53; Brožek, '54), together with the quantitative assumptions on which the equation is based. Some of these assumptions, such as the density of "cell mass," are being currently replaced by more reliable constants. This will result in a modification of the equation given above. For this reason it is preferable to calculate the correlations between partial indicators of body fat and body density, rather than the total fat estimated from a particular densitometric equation.

Care should be taken not to apply the equations predicting density from measurements of subcutaneous fat too readily to individuals differing in sex or age from the sample for which the basic data were obtained. Subcutaneous fat is only a fraction, be it a large one, of the total body fat. It was noted (Brožek, '55, p. 498) that the same skinfold thickness is associated with a higher total fat content in middle-aged men than in young men. An equation derived for the older men would overestimate the total body fat of young men. The difference is due to the different amounts of internal fat. The sex differences in this regard remain to be examined.

Skinfolds vs. roentgenographic values

The spot at the dorsum of the upper arm at which the skinfold was measured was marked with dermatographic pencil and an attempt was made to place the arm in front of the casette in such a way that the site could be located on the roentgenogram. However, as no lead marker was applied in this series, as has been done by Garn and Gorman ('56), and the arm was not clamped in a rigid position, the location was reproduced only approximately on the roentgenograms.

The coefficient of correlation between the thickness of dorsal skinfold, measured on the upper arm, and the corresponding roentgenographic reading for our subjects was 0.824 ($N = 126$), a value that is similar to those available in the literature. Baker ('55) reported $r = 0.852$ ($N = 83$ young adults), presumably for the same site as was used in our study. Hammond ('55, p. 207) obtained r values of 0.82 for the thigh, 0.83 for biceps, and 0.89 for the triceps in a sample of about

150 boys of unspecified age. In Garn's ('56) sample of 65 young men, $r = 0.88$ for measurements made at the levels of the lowest rib at the midaxillary line. On this site the median caliper value of the skinfold (12.0 mm) was only 65% of the radiographic equivalent (2×9.3 mm $= 18.6$ mm). The compression effect is larger, even though presumably the caliper spring exerted the same pressure as in the present study (*i. e.* 10 gm/mm^2) in which the average skinfold value represented 82.3% of the doubled roentgenogrammetric width.

A check was made as to whether the method of measurement (vertical to skin in Garn's work, vertical to the axis of the limb in the present study) would account for the difference between roentgenographic values and skinfolds. The means and S. D.'s for the dorsal width of adipose tissue at the mid-upper arm were 7.90 ± 2.40 mm for the subsample used for correlations with body density ($N = 52$, table 2) and 7.95 ± 2.57 for the total sample ($N = 126$). The corresponding values obtained for measurements made vertically to the skin were 7.86 ± 2.42 and 7.94 ± 2.55, *i. e.* the means were only negligibly smaller. The coefficients of correlation between skinfolds and X-ray widths were 0.824 (table 4) and 0.825, respectively. Thus, at this particular site, the two methods yield essentially identical values and can not account for the differences between Garn's and our data on skinfolds expressed as percentages of roentgenographic fat thicknesses. The problem of regional and age differences in the compressibility of adipose tissue is under study.

SUMMARY

Interrelations between three types of criteria of leanness-fatness (skinfold thickness, width of subcutaneous adipose tissue measured on roentgenograms of the limbs, body density) were examined in 52 men, averaging 57 years in age.

The coefficient of correlation between skinfold measurements at the dorsum of the upper arm and the corresponding roentgenogrammetric values was 0.82.

The average skinfold value, obtained with the pressure of 10 gm/mm^2 of the contact surface of the caliper, corresponded to 82% of the doubled width measured on the roentgenograms.

Equations for predicting body density, used as an indicator of total body fat, were calculated for single sites and for combined lateral-plus-medial or ventral-plus-dorsal roentgenographic measurements.

Relating the width of the layer of subcutaneous fat to stature, bony

width of the extremity, or to the width of bone-plus-muscle has not improved importantly the correlation with body density.

According to the available evidence, the validity of which is limited by the relatively small size of the present sample, measurements made on roentgenograms of the extremities do not appear to be more effective indicators of leanness-fatness than direct (skinfold) measurements. However, a systematic appraisal in terms of multiple regression analysis remains to be carried out.

A comprehensive analysis of the value of measurements made on soft-tissue roentgenograms should include sites on the trunk not accessible to the skinfold-caliper technique. Proper attention should be given to factors of age and sex.

ADDENDUM

When this paper was completed, in the course of a discussion, Dr. E. E. Hunt, Jr. pointed out the potential advantages of using the calculated (approximate) crosssectional areas of the adipose tissue, related to the non-adipose ("lean") tissues of the limbs, as a roentgenographic criterion of fatness. This can be done in the present study for 4 locations in which the skin-plus-fat thicknesses were measured on both sides of the limb (upper arm, half-way up from the olecranon, in medio-lateral projection; upper arm, one-third up, antero-posterior projection; thigh, one-third up from the patella, A-P projection; calf, A-P projection).

TABLE 8

Adipose areas, expressed as percentages of the lean areas in 4 limb cross sections, and the coefficients of correlation of these percentages with body density (r)

LOCATION	MEDIAN	MEAN	S. D.	N	r
Upper Arm, midway	32.6	34.5	11.5	52	—.72
Upper Arm, lower $\frac{1}{3}$	49.6	53.4	14.5	41	—.68
Forearm	24.1	24.3	7.3	51	—.79
Calf	20.2	21.9	7.1	47	—.56

Assuming that the limbs are circular (which, strictly speaking, they are not), the areas of the total crossection and of the "lean" cross-section were calculated from the total and the muscle-plus-bone diameters, respectively. The "adipose" area was obtained by subtraction and expressed as a percentage of the lean area. The descriptive data and coefficient of correlation with density are given in table 8.

The r's are essentially identical with the values obtained by relating the linear measurements of skin-plus-subcutaneous tissue to the "lean" limb width (bone + muscle, see Table 7, column $X/(B+M)$). Only for the Forearm is the r higher than when the absolute value of skin-plus-subcutaneous fat is used, disregarding body size, and even here the difference is trifling (—.76 vs. —.79).

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A COMMENT ON WILBER'S "ORIGIN OF HUMAN TYPES"

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DR. C. G. Wilber has taken the trouble, in the December 1957 issue of *Human Biology*, to refute the notion that simple climatic determinism explains the geographical and local races of man. While rendering a service to the reader who has not followed the periodical *Evolution*, he has, in his article, contributed several errors that bear mention here.

In particular, Wilber is concerned with the inadequacy of Allen's and Bergmann's "rules," as they pertain to homeotherms in general, and especially to *Homo*. As examples of exceptions he says that "the hippopotamus, rhinoceros and elephant are built on the roly-poly, globular principle, but are well entrenched in their tropical environments" (Wilber, '57, p. 332). At least two of these three examples possess unique adaptations, one of them (the elephant's ears) definitely increasing the body surface to an important degree, and the second (the hippopotamus' aquatic habits) serving to immerse the animal in coolant, thus extending the surface area indefinitely.

However, it is in regard to the Eskimo, a group with which Wilber has personal experience, that serious errors are made. Quoting Jenness, he writes that "with respect to *all* Eskimos" (italics are mine) "they incline toward slenderness, the body tapering a little below the broad shoulders" (p. 333).[1] And, as evidence of this generalization, he cites Ray's 1885 height-weight data on Point Barrow males and females. These data, however, yield little proof of slenderness. In both sexes the Point Barrow weight-height ratio is high. Ray's Point Barrow females, though five inches *shorter* than Ohio white females (Garn, '57a), still averaged two pounds *heavier*. If these figures bear quoting, they strongly support the assumption of a high mass/volume ratio for Eskimos!

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[1] Steller, on the other hand, described the Aleut as "of medium stature, strong and square-built with muscular arms and legs. The neck is short and the shoulders broad." (Steller, 1793).

Similarly, among Aleut children, the weight-to-height ratio is distinctly high, as Wilde ('50) has shown for the St. Paul Aleut, and C. F. A. Moorrees and I have demonstrated for the Attu-Atka-Umnak Aleut (Garn and Moorrees, '51). Hrdlička's data on Kuskokwin Eskimo children may also be cited (Hrdlička, '41). As data both on Aleut children and on adult Aleut show (Laughlin, '49), there is a pronounced reduction in the long bones and peripheries, a trend in the expected direction, if it is assumed that reduction of the peripheries may be a genetic adaptation to a cold climate.

Wilber also makes much of the facts that many Arctic-dwelling people keep their homes at an elevated temperature, quite uncomfortable for us, and possess the best-engineered clothing that existed prior to the development of the deep-sea diver's dress. The latter, at least, is obvious. One must be equipped for the cold to exist in it (*cf.* Coon, Garn and Birdsell, '50, pp. 66-67). The former becomes irrelevant, as soon as we remember that cold-selection operates out-of-doors, on the hunt, driving sledges, when feeding reindeer, or even at quite moderate temperatures, paddling a thin-bottomed kiyak from Atka to Attu. It is out-of-doors rather than snug at home that differential survival of the shorter-limbed, better-padded Eskimo may have contributed to the high weight-height ratios and shorter limbs that persist today.

It is not to be expected, however, that generalizations are any more than generalizations, and when we encounter a human population repeatedly exposed to some potentially selective factor, it becomes imperative to look for multiple adaptations, using experimental techniques as Wilber suggests.[2] At least two differences, probably indicative of genetic and not physiologic adaptations are known for Southampton Eskimos. One is the maintenance of higher peripheral skin temperatures, under conditions of cold-water immersion (Brown and Page, '52); the second, and more puzzling, is the maintenance of lower intramuscular temperatures under the same conditions (Brown, Hatcher and Page,

[2] Here a quotation from Volpe is pertinent. "In many studies on evolution, there is a tendency for investigators, of which the writer claims to be no exception, to interpret, or rather 'fit,' their data to prevailing evolutionary theories, rather than to design experiments to test critically the validity of the current postulates or to exploit new possibilities." (Volpe, '58, p. 367).

'53). These are not quite "typical" tropical adaptations but suggest the possibility of a complex of adaptations to cold.

However, as I have pointed out before, the critical test is not the simple possession of possibly-useful adaptations, but rather differential survival (Garn, '57b, p. 220).

I thoroughly agree with Dr. Wilber that man is primarily a heat-adapted animal, as performance experiments and man's unique sweat-gland system both attest. He is, moreover, a speed animal, a rather lightly-muscled collection of folding bones, to paraphrase Christopher Fry. Nevertheless there are indications in the way body size and body proportions are distributed about the world, that some generalizations pertinent to other homeotherms do apply to man (*cf.* Roberts, '53). Despite air-conditioning and central heating, the majority of men still venture out-of-doors, and thus subject themselves to temperature selection.

I know of no anthropologist so rash as to claim that temperature and the radiant heat load are exclusive or even major causes of the differences between geographical races, or that the past-century formulations of Bergmann and Allen completely solve the problems of race formation in man. I know of none who adopt the Lamarkian approach that Dr. Wilber so gleefully demolishes. But, when Wilber asserts that "the rules of Bergmann and Allen have *no* causal role in the formation of racial differences in man" (p. 335), I doubt very much that he intended such a sweeping and untestable counter-generalization.

NOTE

In regard to climatic adaptations, there are several recent articles. It is not entirely clear, however, whether they all score a point for Wilber, or for the present author.

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BOOK REVIEWS

Principles of Research in Biology and Medicine. By DWIGHT J. INGLE. xv + 123 pp. Lippincott, Philadelphia, 1958. $4.75.

Professor Ingle, well-known physiologist associated at present with the Ben May Laboratory for Cancer Research, University of Chicago, has been one of the moving forces behind the establishment of the new journal *Perspectives in Biology and Medicine,* designed to facilitate communication of ideas among scientists. He has been disturbed, as were others, by the danger growing from the steadily rising pressure for space in scientific journals, resulting in editorial policies that emphasize the most concise presentation of data and limit severely historical considerations of the problem and the discussion of the results.

While to Ingle the history of biological science is essentially the history of great hypotheses and their protagonists, he is no advocate of uncritical thinking or armchair speculation. As an experimentalist, Ingle is well aware of the restrictions that must be placed on ideas. Experimental evidence, and this includes a proper statistical analysis of the data, is the unquestioned court of appeal, the experimental scientist's only genuine criterion for the discrimination between fact and fancy. Yet, experiment *and* theory are inseparable parts of scientific inquiry. Answers do depend, in part, on questions we ask and the synthesis of data (as differentiated from a mere summation of isolated facts) requires perspective, i. e. theory. The choice of worthy goals is the most difficult problem facing the scientist, once the technical tricks of the trade had been mastered and material opportunities for investigative work had been secured.

The *Principles* are an expression of Professor Ingle's concern about the more general (philosophical) aspects of research, aspects he finds entertaining personally and considers, with justification, as being commonly neglected in scientific education. The creative scientist differs in important ways from the skilled but unimaginative technician, even though the essential differences are subtle, hard to analyze adequately and, consequently, at the present time, beyond control and transmission by educational methods. "It is no more possible," says the author, "to teach a scientist how to make a great discovery than it is possible to teach a poet how to write a great poem, an artist to paint a great picture, or a composer to write a great symphony" (p. viii).

Yet, in addition to factual information and manipulative skill that must be acquired by the student of a particular biological discipline or medical specialty, there are general principles concerning the gathering of data ("experimental design") and their interpretation that *can* be taught. The present book is the fruit of the conviction that these principles *should* be taught.

A large part of the volume is devoted to spelling out the difficulties inherent in establishing a fact. The unaided perceptual organs of man are sensitive only to a small proportion of energy manifestations, and they are incapable of precise quantitative appraisal even when the stimuli fall within the sensory range. Consequently, much of the scientist's effort goes into development of physical aids for the detection and measurement of natural phenomena. The scientist should be aware of the seductive effects of suggestion, frequently subtle, upon his perception and thought processes. Bias must be avoided in the sampling of populations under study and in the execution of experiments.

Defining a cause of any specific event as "any necessary antecedent or concomitant event or condition" focuses attention upon the complexity of "causes" and the importance of concomitant conditions. In addition to the relatively stable framework of "necessary conditions," the author lists several categories of "permissive" (supporting, secondary) causes and analyzes the "active" causes as variables that a) trigger the response of a system already organized for response, b) facilitate a reaction as catalysts, or c) induce response by transmitting energy directly to the system.

There is an interesting discussion of "heteropoietic" (difference producing) factors, the naturally occuring extrinsic and intrinsic variables (such as species differences and genetically determined biochemical individuality, sex, age, activity, emotionality, characteristics of the physical environment, food intake, physiological rhythms, and adaptation) which may affect responses to a given experimental regimen. The last chapter deals with "Relationships among Scientists," both at the educational level and in reference to various forms of organized research.

Throughout it is stressed, for the benefit of the experimenter and the clinical investigator, that statistical methods are of increasing importance in the design of experiments as well as in the interpretation of the data. The two aspects, design and analysis, are intimately connected. No claim is made that formal statistical analysis is an infallible guide to decision or that it replaces common sense. Neither the earth-bound training in specific research techniques nor an exposure to the more rarified atmosphere of philosophy of science can provide a sure guide for dealing successfully with the unknown—the goal of all "fundamental" research. But awareness of the general principles of research should usefully widen the student's perspective and sharpen his criteria of scientific proof.

The present volume is the handiwork of a thoughtful scientist, not of a professional philosopher of science. On the whole, this is all to the good. It is reflected in the effective use of references to problems encountered in actual investigative work, and in the addition of a welcome and healthy psychological element to the purely logical considerations of "truth and consequences." Thus enjoyment of inquiry is regarded as such an important ingredient of scientific investigation that it is listed, together with understanding of natural phenomena and their prediction and control, among the general aims of science: "Research should be fun. It should be possible for the scientist to find enjoyment in the laboratory and in the fellowship of scholars throughout his life." And again: at the introductory level, "science should be presented in all its color, beauty and drama, indeed, the exciting qualities of science should not be lost from graduate education or from life in the laboratory, although discipline and attention to detail is also required."

This is not a systematic *Handbuch* and was never intended to serve as such. Readers will vary in regard to the items they should like to see treated in greater detail. Here are some topics that might be considered in the next edition: Financial aspects of research, including the benefits and limitations of contract research. Problems resulting from increasing cost and complexity of instrumentation. Effects of the steadily rising flood of publications and the new developments in the "retrieval" of scientific information. The growing disproportionality between periods of "inspiration" and "perspiration" in scientific research. The potential conflict, in individual scientists, between the devotion to science—the all-demanding mistress—and personal responsibilities to one's family and the community.

There are points that could be criticized, such as topical sequence or the unevenness of style. Some parts read like well written essays. Others resemble lecture outlines, and some ("Basic assumptions of science," p. 4; "Fallacies," p. 60) are little more than inventories of titles.

However, the reviewer's criticisms concern only the periphery. The core is solid, stimulating and worthwhile—not only for the intended audience of research workers in their fetal or neonatal state but also for the seasoned veteran. While Ingle wrote for students, he expressed a conviction that the mature investigator may profit at times from looking at his own handiwork from a greater distance. In fact, he should be a student of the philosophy of research throughout his life.

The reviewer is convinced that more than one reader—at some point or another in the perusal of the slim volume—will respond like one of Ingle's favorite heroes of detective fiction: "It makes one furiously to think." Can there be a greater compliment?

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Changes in Body Water Compartments During Growth. By BENT FRIIS-HANSEN. ii + 68 pp. Ejnar Munksgaard, Copenhagen, 1955. (Acta Paediatrica Supplement 113).

Here is an important description of the relative loss of body water in children during growth. It was written by one of the most active investigators of the subject, and stems from years of collaborative work carried out both in Boston and in Copenhagen. This and other recent studies have provided considerable data on the growth of body fluid compartments in infants and children, but published information on adolescents is still scarce. Furthermore, the physiological mechanisms which regulate these changes are still very poorly known.

Friis-Hansen begins his monograph by describing the observations of ancient Greek and Roman writers on the relatively high moisture content of human infants. He then reviews the methods and hypotheses underlying the measurement of body water in the living, and defends his procedures as among the more practicable for work on the young. The dilution of deuterium oxide in the blood after injection is his estimate of "total" body water: while the comparable dilution of thiocyanate approximates the "extracellular" fraction of body fluids. The difference between these two "spaces" is taken as the "intracellular" water.

This review is adequate except for a curious lack of references to recent work on the density and hydration of the "fat-free" body—particularly the research at Minnesota summarized most fully by Keys and Brožek in 1953 (Physiological Reviews *33*:245-325).

Friis-Hansen's data pertain to 62 boys and 31 girls, ranging from neonates to a few teen-agers. For some of these children, both deuterium oxide and thiocynate determinations were completed, but for most, only one of the two measurements was carried out. The sample came from hospitals, but was evenly distributed above and below recent norms of stature and weight as related to age. The norms used were the closely similar ones for Denmark and Boston.

Friis-Hansen's statistical treatment consists of regressions of the logs of weight, stature, and body fluid compartments on the log of age from birth. He also makes use of surface area calculated from stature and weight. For each constituent, he separates his combined sample of both sexes into 5 age-groups, and has computed 5 linear regressions.

The logarithmic scale of age from birth is awkward in that it is so greatly stretched in the first few postnatal days. This difficulty is avoided by adding an interval equivalent to the average human gestation period, so that the scale approximates the log of conceptional age (*cf.* Hunt *et al.*, 1958, Human Biology *30*: 73-91). Even this device, however, only slightly reduces the number of regressions of log measurements on log conceptional age which seem to be indicated by the data. The allometric relationships between the constituents, however, are far more regular and simple, as Friis-Hansen and others have shown.

On the basis of his regressions on small age-samples, Friis-Hansen assumes that no sex difference exists in the growth trends of his measurements. Most large-scale growth studies in the literature, however, indicate a true sex difference in weight—especially in pre-school and adolescent samples. It would appear from the evidence, however, that the true sex difference in body *composition*, as opposed to weight alone, is inconsequential before pubescence.

Friis-Hansen's most important findings are that in the first postnatal year, the percentage of extracellular fluid in the body decreases while the intracellular fraction stays fairly constant. In the second year, both fractions increase—very likely along with a decrease in total body fat. Later on, the extracellular compartment continues its decrease: while the intracellular fraction show only minor changes. These trends seem to reflect the increasing cellularity of the growing body, with a concomitant relative decrease of extracellular fluid.

To sum up: the weaknesses of this monograph are minor, and it is a most satisfying example of Scandinavian workmanship in human biology.

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EDITOR'S NOTE: Two other important monographs, neither of which was available to Friis-Hansen, were later published on the subject of body composition: 1) Human Standard and Maximal Metabolic Rate in Relation to Fatfree Man. By Wilhelm von Döbeln. 79 pp. Stockholm, 1956. Acta Physiol. Scand. *37*, Suppl. 126, 2) The Gross Composition of the Body. By William E. Siri. Adv. Biol. Med. Physics, *4*: 239-280, 1956. Cf. also Evaluating Body Composition. By George R. Cowgill. Borden's Rev. Nutritional Res. *19* (No. 1): 1-17, 1958.

Heights and Weights of Children and Youth in the United States. By MILICENT HATHAWAY. 131 pp. Home Economics Research Report No. 2. U. S. Department of Agriculture. U. S. Government Printing Office. 1957. $0.65.

Standard of heights and weights are useful to many disciplines concerned with child health. But where to find suitable standards has been a constant worry. Doctor Hathaway has done a great service by collecting the best available in the United States. Most importantly, she has emphasized the source of the data, with particular reference to date of collection and the geographical location of the children concerned. How often are we guilty of comparing to-day's child born and bred in X state with standards compiled in 1920 in Y state? The compilation is limited to height and weight data on white boys and girls from 2 to 18 years. How lamentable it is that well collected data on the Negro child are precious and rare; he provides a large measure of child health problems in this country.

The first part consists of general tables from studies in 34 states and the District of Columbia. Classification is by region, State and date of study, and at the end of each table of a certain age there are admirable and purely arbitrary standards for that age. These are considered the best available data. To give an idea of the comprehensive nature and labor of Doctor Hathaway's work, there are, for example, 68 sets of data listed under 9 year old boys.

The second section presents selected height-weight standards from different sources and examples of forms and graphs in use. It was good to see, for comparative and historical interest, that Hastings' much neglected and admirably collected data of 1889 were given. This section is valuable because it deals with the distribution of the measurements in the samples and affords some idea, usually by percentiles or standard deviation, of the range of "normality." A most curious fact can be seen on studying the Jackson-Kelly growth charts where, very properly, percentiles are used for 'channels' of growth in weight, and standard deviations for height: in several places the curves of plus or minus 1 standard deviation from the mean are clearly not equidistant from the mean curve. This discrepancy has found its way into some of the corresponding table and there are therefore some plus 1 standard deviations which are different to the corresponding minus quantities—clearly an impossibility.[1] Perhaps the original calculations of the deviations were not traditional. This most interesting section is enhanced by the author's descriptive notes heading each set of data.

The third section deals with some factors affecting growth patterns and here such variables as nationality, socio-economic status, decade of measurement, and rate of maturity are plotted to show their effects. This last factor—rate of maturity—affords the necessity to write a note of sadness: the presentation of Doctor Nancy Bayley's most important curves of the growth of children maturing at different rates are the only ones giving incremental or velocity data in the entire vast collection. Sad because many of us need velocity standards so badly, and because the presentation of longitudinally collected data cross-sectionally is hardly getting the most out of hard invested work. It need hardly be said that not one tittle of criticism is directed here at Doctor Hathaway—the published data were not there for her to seek and find.

Finally, the last part consists of an annotated list of literature references. It is comprehensive and is made doubly useful by the excellent annotations.

This paper-backed report should certainly be in the hands of all students of growth and development.

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[1] Doctor Hathway is now aware of these discrepancies and corrections are available.

The Numbers of Man and Animals. Edited by J. B. Cragg and N. W. Pirie. 152 pp. Macmillan, New York, 1957. $2.75. (Originally published by Oliver and Boyd, London, 1955).)

This small volume contains the very stimulating record of a symposium held in England in 1954 by the Institute of Biology. The objective of the conference was to assemble specialists from the wide variety of sciences which in some way are concerned with the growth of animal and human populations, in order to discover how much common ground existed and to highlight gaps in our knowledge. The orientation of the volume is well expressed by the editors: "The growth of human populations is strongly influenced by social and cultural factors which are much less important in other species. For all that, *Homo sapiens* is an animal and many aspects of his relationship with his environment, with other species, and with other members of his own species, should be open to biological analysis."

The book contains 13 all too brief contributions by illustrious specialists in economics, demography, cultural anthropology, mathematics, biology, human ecology and other disciplines, most of which are followed with only occasionally provocative discussions. The papers range over such topics as an economic theory of optimum population, the effects of nutrition on fertility, illustrative mathematical models of population dynamics, research in the biology of conception and the future possibilities of telegenesis, experimental competition in beetles, and glimpses of the future population and natural resources of the world.

To any reader who is a specialist in any area covered, the contributions from that area, with few exceptions, will probably seem quite familiar. But for most members of the natural and social science fraternities, at least a third and probably more of the selections will provide both new information and fresh points of view. One cannot help but be profoundly impressed with the number and diversity of important perspectives brought to bear on the subject of population growth. For anyone interested in this subject, this little book is a genuinely educational experience. The only negative reaction stems from having one's appetite whetted and so quickly frustrated by the brevity of the offerings.

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Mastery of Stress. By D. H. FUNKENSTEIN, S. H. KING and MARGARET E. DROLETTE, Harvard University Press, Cambridge, Mass., 1957.

The book reports the results of a two-year investigation carried out with men attending Harvard College. The title is rather misleading, in that mastery and stress are dealt with only within the narrow confines of distinctly limited experimental conditions: performing arithmetical problems (with some "heckling") and reading a story under conditions of "sonic confusion" in which the subject heard, by means of earphones, his own voice with a slight delay. The main variables were as follows: characteristics of sequential reaction to repeated presentation of the "stress" situations (i. e. in the authors' words, whether "stress was mastered"), physiological reactions and patterns, direction of "emotional" reaction (e. g. "anger-in," "anger-out," "anxiety"), and perception of parental roles. With reference to the last item, the investigation represents a most commendable pioneering effort relating perception of parental roles to physiological reactions and performance.

The main purpose of the physiological measurements was to test the hypothesis that—contrary to Cannon's thesis—physiological reactions do actually differentiate between different emotional states, that—for example—anger-out is a nor-epinephrine reaction and that anxiety is an epinephrine reaction. It is a very questionable point in this reviewer's mind whether the amount of emphasis given to this dichotomy between epinephrine and nor-epinephrine is justified by the data or by the heuristic value of such a distinction. In any event, considerably more physiological data are required before concluding that physiological reactions furnish generally reliable means of differentiating anger and fear.

Wholly apart from the matter of the reason for including physiological measures in the present study, the physiological part of the investigation may be criticized for omitting some key physiological indicators (such as palmar conductance and respiration) that may well have produced results leading to different conclusions. The actual choice of indicators appears arbitrary. The authors admit that their interpretation of the exact physiological mechanisms lying behind the ballistocardiographic tracings is highly conjectural, and they mention the difficulty that clinicians have "in getting the same tracings on the same patients on repetition," suggesting that the inconsistencies may be due to "shifts in the patients' emotional reaction to the testing situations at different times." However, such inconsistencies could very possibly be due to inherent unreliabilities in the measurement process itself. Actually, estimations of reliability are not given for methods employed in the present study.

In my judgment, one of the most serious weaknesses of the book is that although it is amply discursive, the authors do not appear to seek any manner of satisfactory nexus with current psychological theories. References to such theories are limited to writings of nearly two decades ago. The term "emotion" is used repeatedly without any reference to recent psychological papers dealing with this topic, and to closely related topics such as that of motivation. Indeed, it is surprising that the term "motivation" does not even appear in the index. Crucial terms ("anxiety," "depression," "stress," "conflict," "frustration") are not defined with sufficient rigor, in my opinion. Moreover, in some instances, terms denoting particular emotions are used interchangeably with terms denoting diagnosis of complex psychopathological states.

Despite its defects and omissions, this study represents an important piece of investigative work. Its importance does not lie mainly in the conclusions permitted by the findings, although there are a considerable number of suggestive findings. Rather, this work—as I see it—is important for its originality, as a long step forward into a new research area. It is to be hoped that the authors will continue along these fruitful lines.

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Verbal Behavior. By B. F. SKINNER. x + 478 pp. Appleton-Century-Crofts, New York, 1957. $5.50.

Man's ability to verbalize is his most advanced and unique form of behavior. The outstanding contribution of Skinner's book is to offer a framework in which man's verbalizations can be examined with the same objectivity and scientific rigor as other aspects of his behavior. Skinner views previous attempts to systematize the terminology describing verbal behavior as failing to provide the techniques necessary for a causal analysis of human speech. He believes that approaches such as semantics, logic, grammar, and rhetoric are essentially dualistic in their conceptualizations. Terminology such as "ideas," "images," and "meanings" imply a "mentalistic" or inner component in man which is distinct from his overt and observable behavior. In contrast, Skinner postulates that the lawful relationships embodied in speech are the same as those governing all other human behavior. In his experimental work, Skinner has gone further than any other scientist in eliminating the "mind-body" dichotomy.

Skinner conceives of this book as being an exercise in "interpretation" rather than an extrapolation of previous experimental results. Thus he deliberately avoids citing the psychological literature in this area. From the viewpoint of the research specialist, it is unfortunate that Skinner limited himself to the briefest mention of an ever-increasing body of data which support his approach to verbal behavior. However, the general reader will find that his procedure of using literary and philosophical writings as his primary data greatly enhances the readability of the book.

For those readers who may be unacquainted with Skinner's views about man, science, and society, it is suggested that this book be read in conjunction with two of his previous works, "Science and Human Behavior" and "Walden Two." The latter is a science fiction version of the ideal society based on Skinnerian conditioning principles. As in these previous volumes, Skinner is saying that under the proper conditions all human behavior, including verbalization, is understandable, predictable, controllable, and manipulable. In this age of hidden persuaders," "subliminal perception," and "brainwashing," a book which deals with the lawful effects of one person's behavior upon that of another person must have important consequences.

It should be pointed out that this book has been long anticipated. Skinner has presented his basic views about verbal behavior in university courses at Minnesota, Harvard, Chicago, and Columbia, and mimeographed material from these courses has been circulating for the past ten years among interested researchers. Skinner's influence on American psychology is increasing at a rapid pace. If one can judge the importance of a book in a scientific field by the amount of research it generates, then it is highly probable that this book will be a scientific landmark.

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BOOK NOTICES

Introduction to Biostatistics. By Huldah Bancroft. x + 210 pp. Paul B. Hoeber, New York, 1957. $5.75. Developed in the course of teaching biostatistics to sophomore medical students at Tulane University. In addition to the usual materials, short chapters on life tables and bioassay are included.

General Systems, Vol. II. Edited by Ludwig von Bertalanffy and Anatol Rapoport. xiii + 188 pp. Yearbook of the Society for General Systems Research (R. L. Meier, University of Michigan, Ann Arbor, Mich., Secretary), 1957. No price indicated. The second Yearbook continues to provide a forum for a variety of approaches, reflecting the fact the interests in the problem range from preoccupation with mathematical models to philosophical speculation, with emphasis on human behavior as subject matter.

Anatomist at Large. By George W. Corner. v + 215 pp. Basic Books, New York, 1958. $4.00. An informal but informative autobiography (pp. 3-64), followed by essays ranging from technical topics (Physiology of the Corpus Luteum) to philosophy and history of medicine. Appropriately, the author upon retirement as Director of the Department of Embryology of the Carnegie Institution became the historian of the Rockefeller Institute for Medical Research.

Evolution of Genetic Systems. By C. D. Darlington. x + 265 pp. Basic Books, New York, 1958. $5.50. While the title is the same as that of the 1939 edition, the present volume is essentially a new book, motivated by the purpose "to join together the relatively fixed world of physics and chemistry and the undoubtedly moving world of biology," "to see how the shape of our knowledge is being changed today just as it was changed in the past by the work of Mendel, Darwin and Pasteur."

Science and the Creative Spirit. By Karl W. Deutsch, F. E. L. Priestley, Harcourt Brown and David Hawkins. xxvii + 165 pp. University of Toronto Press, Toronto, 1958. $4.50. Four essays on humanistic aspects of science. "While this book results from an effort to reduce the area of disagreement betwen humanist and scientist, it does not seek that end by denying that conflict exists."

Aged in American Society. By Joseph T. Drake. ix + 431 pp. Ronald Press, New York, 1958. $5.50. Presentation of gerontology in sociological framework. Part IV deals with physiosomatic and psychosocial characteristics of the aged.

Darwin's Century. By Loren Eiseley. xvii + 378 pp. Doubleday, New York, 1958. $5.00. A vivid portrait of "evolution and the men who discovered it."

CHEMICAL ANATOMY, PHYSIOLOGY AND PATHOLOGY OF EXTRACELLULAR FLUID. By James L. Gamble. 164 pp. Harvard University Press, Cambridge, Mass., 1958. $2.25. Sixth edition, attesting the continued usefulness of this lecture syllabus.

FUNDAMENTALS OF NEUROLOGY. By Ernest Gardner. xi + 388 pp. W. B. Saunders, Philadelphia, 1958. No price indicated. Third edition, revised with special attention to sensory endings in muscle, to vision, and to basal ganglia.

FADS AND FALLACIES IN THE NAME OF SCIENCE. By Martin Gardner. x + 363 pp. Dover Publications, New York, 1957. $1.50. A revised and expanded edition of this study in human gullibility, reviewing "the curious theories of modern pseudoscientists and the strange, amusing and alarming cults that surround them."

CURRENT TRENDS IN THE DESCRIPTION AND ANALYSIS OF BEHAVIOR. Edited by Robert A. Patton. 242 pp. University of Pittsburgh Press, Pittsburgh, 1958. $4.00. A series of 9 papers on topics ranging from psychophysiology to psychoanalysis, focussed on the problem of description—preferably quantitative—of behavioral phenomena.

INDUSTRIAL PSYCHOLOGY. By Thomas Willard Harrell. xiv + 398 pp. Rinehart & Company, New York, 1958. $6.00. Revised edition of a college textbook, first published in 1949, emphasizing the concept of motivation.

JOB ATTITUDES: REVIEW OF RESEARCH AND OPINION. By Frederick Herzberg, Bernard Mausner, Richard O. Peterson and Dora F. Capwell. xii + 279 pp. Psychological Service of Pittsburgh, Pittsburgh, 1957. No price indicated. Beginning with the problems of the prevalence of job dissatisfaction and the characteristics of dissatisfied workers, the authors critically review the voluminous literature on different aspects of job attitudes, ending appropriately with the considerations of mental health in industry.

GROWTH OF LOGICAL THINKING FROM CHILDHOOD TO ADOLESCENCE. By Bärbel Inhelder and Jean Piaget. xxvi + 356 pp. Basic Books, New York, 1958. $6.75. Monograph on the development of problem solving behavior, with emphasis on the period from 11 to 15 years, crucial for the study of the transition from the concrete thought of the child to the formal thought of the adolescent.

MANUAL FOR NUTRITION SURVEYS. Interdepartmental Committee on Nutrition for National Defense. viii + 160 pp. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 1957. $1.50. Designed "to serve as a guide for survey teams in collecting data for an assessment of the nutritional status of a population." Includes measurement of skinfold thickness (p. 29).

PHYSIOLOGY OF MAN. By L. L. Langley and E. Cheraskin. x + 674 pp. McGraw-Hill, New York, 1958. $6.95. Second edition of an introductory text, "dedicated to the proposition that learning can be fun."

PRESERVATION OF YOUTH. By Moses Ben Maimon. Transl. by Hirsch L. Gordon. 92 pp. Philosophical Library, New York, 1958. $2.75. Written in 1198 for the Sultan of Egypt and now translated from the Arabic original, the essays provide an interesting picture of the medieval views on various aspects of the medieval curative and, in particular, preventive medicine.

HUMAN ENGINEERING. By Ernest J. McCormick. xi + 467 pp. McGraw-Hill, New York, 1957. $8.00. "Human engineering," concerned with the principles underlying the rational adaptation of equipment, work space, and work environment for human use, is essentially an interdisciplinary enterprise, with contributions from engineering, physical anthropology, medicine, physiology, and psychology. The book covers the field with emphasis on the contributions of psychology.

CURRENT STUDIES IN PSYCHOLOGY. By F. J. McGuigan and Allen D. Calvin. xiii + 226 pp. Appleton-Century-Crofts, New York, 1958. $2.65. A useful presentation of selected recent (since 1950) papers in several fields of psychology (learning, motivation, growth and development, personality, perception, social factors), made accessible to beginning undergraduate students.

BIOGRAPHICAL MEMOIRS, Vol. XXXI. National Academy of Sciences. vi + 396 pp. Columbia University Press, New York, 1958. $5.00. Includes, among others, biographies of E. G. Conklin (biologist), W. S. Hunter (psychologist), R. Linton (anthropologist) and J. P. Peters (biochemist).

DECENNIAL SUPPLEMENT (ENGLAND & WALES, 1951)—AREA MORTALITY. By the General Register Office. xxviii + 82 pp. British Information Services, New York, 1958. $12.50. Mortality statistics contribute to biochemical knowledge in terms of changes over time, differences between social and occupational groups, and differences between regions. The present volume presents death rates for geographical subdivisions of England and Wales during the period 1950-1953.

TISSUE ELASTICITY. Edited by John W. Remington. vii + 203 pp. American Physiological Society, Washington, D. C., 1957. $4.50. The visco-elastic properties of living tissues are of interest to physicists and chemists (protein structure and elasticity) as well as to biological scientists studying elastic properties of muscles, blood vessels and lungs. W. O. Fenn considered elasticity in the human body (p. 98). A useful glossary of terms (p. 191) is appended.

METABOLISM OF THE NERVOUS SYSTEM. Edited by Derek Richter. xiv + 599 pp. Pergamon Press, New York, 1957. $16.00. Proceedings of the Second international Neurochemical Symposium, held in Denmark in 1956. The topics range from electronmicroscopy of nervous tissues to transmission in the central nervous system and drug action.

THE CHILD: A BOOK OF READINGS. Edited by Jerome M. Seidman. xiv + 674 pp. Rinehart & Company, New York, 1958. $6.75. Collection of writings, presented in the original form, on various facets of child development, from infancy to adolescence. Three (out of 65) papers deal with neuromuscular growth.

MEASUREMENT AND STATISTICS. By Virginia L. Senders. xvi + 594 pp. Oxford University Press, New York, 1958. $6.00. "A basic text emphasizing behavioral science applications."

COMBINATION OF OBSERVATIONS. By W. M. Smart. xiv + 253 pp. Cambridge University Press, New York, 1958. $6.50. The treatment of observations and measures subject to errors constitutes the core of this volume. Of special interest are chapters 2 (Errors of observation), 5 (Measures of precision for weighted observations), and 8 (Correction of statistics).

HANDBOOK OF BIOLOGICAL DATA. Edited by William S. Spector. xxxvi + 584 pp. W. B. Saunders, Philadelphia, 1956. $7.50. This is the third publication resulting from an ambitious project to gather and compile, in handbook form, basic established data in the biological and medical sciences (Standard Values of Blood, 1952; Standard Values in Nutrition and Metabolism, 1954). A large number of the tables refer to man's biochemical characteristics, genetics and reproduction, development and morphology, nutrition, respiration and other physiological functions, environment and survival, and vital statistics.

UNDERSTANDING HUMAN MOTIVATION. Compiled and edited by Chalmers L. Stacey and Manfred F. DeMartino. xv + 507 pp. Howard Allen, Inc., Cleveland, 1958. No price indicated. The authors brought together 42, well selected, slightly abridged articles by authors of varied theoretical orientation, to provide a readable introductory text on the dynamics of human behavior.

PERSON PERCEPTION AND INTERPERSONAL BEHAVIOR. Edited by Renato Tagiuri and Luigi Petrullo. xx + 390 pp. Stanford University Press, Stanford, Calif., 1958. $7.50. Outcome of a symposium, held at Harvard University with the support of the Office of Naval Research, concerned with the ubiquitous problem of how we know and what we know about others.

NONPARAMETRIC AND SHORTCUT STATISTICS. By Merle W. Tate and Richard C. Clelland. ix + 171 pp. Interstate Printers and Publishers, Inc., Danville, Ill., 1957. No price indicated. Intended as a manual for graduate students and research workers in social, biological and medical sciences.

PERSONALITY: AN INTERDISCIPLINARY APPROACH. By Louis P. Thorpe and Allen M. Schmuller. v + 368 pp. Van Nostrand, Princeton, N. J., 1958. $5.50. Attempt at an introductory synthesis based upon the conviction that the study of personality must cut across many disciplines and draw upon psychology, psychoanalysis, sociology, education, social psychology and cultural anthropology.

ÜBER DIE ERBLICHKEIT DES NORMALEN ELEKTROENZEPHALOGRAMMS (On the inheritance of normal electroencephalograms). By F. Vogel. 92 pp. Georg

Thieme Verlag, Stuttgart, 1958. $2.30. The monograph is based on 208 pairs of twins, 6 to 30 years of age. The e. e. g. was taken during sleep, at rest and under the stress of hyperventilation and oxygen deficit.

BIOCHEMISTRY AND HUMAN METABOLISM. By Burnham S. Walker, William C. Boyd and Isaac Asimov. vii + 937 pp. Williams & Wilkins, Baltimore, 1957. $12.00. Third edition, keeping up with the advancements in a rapidly expanding field (including complete chemical analysis of a fresh adult human cadaver, in section on Tissue Chemistry). A large section (pp. 404-444) is devoted to the biochemistry of reproduction and heredity.

PERCEPTUAL DEVELOPMENT. By Seymour Wapner and Heinz Werner. 95 pp. Clark University Press, Worcester, Mass., 1957. No price indicated. The monograph deals with changes in spatial organization that takes place in the course of human growth, considered within the framework of a theory of perception which takes into account both the object-stumuli (psychophysical aspects) and the state of the organism (psychophysiological aspects).

PSYCHOTHERAPY BY RECIPROCAL INHIBITION. By Joseph Wolpe. xiv + 239 pp. Stanford University Press, Stanford, Calif., 1958. $5.00. The proposed theory of neurosis and the method of therapy stem directly from modern learning theory. It is offered as a serious alternative to the repression theory and the therapeutic practices of psychoanalysis which stresses the uncovering and expressing of " the repressed."

MEASUREMENT OF COLOUR. By W. D. Wright. ix + 263 pp. Macmillan, New York, 1958. $10.75. Up-to-date presentation of the principles, methods and applications of the trichromatic system of color measurement.

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Minneapolis, Minnesota

JOSEF BROŽEK

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INFORMATION FOR CONTRIBUTORS

HUMAN BIOLOGY, a record of research, is a quarterly journal devoted to the subject its name implies. It publishes original articles dealing with human genetics, development, growth, reproduction and aging, functional anatomy, bio-anthropology, human ecology, population dynamics, etc.

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